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Title	Artificial pneumothorax in the treatment of pulmonary tuberculosis
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Qualification	MD
Year	1921

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THESIS for M.D. 1921.

ARTIFICIAL PNEUMOTHORAX IN THE TREATMENT OF PULMONARY
TUBERCULOSIS.

With Notes on 20 Cases so treated.



INTRODUCTION.

Probably it has been realised from the earliest medical times that the best treatment for an inflamed tissue or part is rest for that tissue or part. It is only of comparatively recent history, however, that the true value and definition of rest - that is absolute physiological rest - has come to be realised, and perhaps in the treatment of no disease has its value as a therapeutic agent come to be realised, as in tuberculosis - more especially so-called surgical tuberculosis of bones and joints. To a large measure this so-called expectant measure of rest has, if not actually taken the place of, made active surgical interference unnecessary in many cases, which in former days would have been unhesitatingly treated by active surgical measures.

Turning to tuberculous disease of the lungs, a new problem faces us. While with reasonable care, it is possible by means of extension, plasters, splints, etc. to give a tuberculous joint the necessary rest to favour its recovery, to control the movements of the lung is not such a simple matter. For here is a tissue forming an organ, which is in constant activity and motion day and night, making on an average twenty excursions per minute - nor can

2

these movements, with the exception of a second or two, be controlled by the will, but are determined by such intricate mechanism as the carbon dioxide content of the blood acting on the respiratory centre, and the intra-pulmonary pressure acting on the vague nerve endings in the lungs. It is clear then that none of the ordinary measures will secure rest for the lung tissue, nor can we expect to influence for any length of time these factors which normally and physiologically control the movements of the lung.

The problem therefore set before us has come to be this - under what conditions during life does the lung tissue cease to expand and collapse? and the answer comes back when the lung is compressed by something, be it fluid or gas in the pleural cavity exerting a mechanical compressing force on the outside of the pulmonary alveoli and not permitting these to expand. Thus it is that in a pleurisy with a large effusion or in a pneumothorax - whether due to a perforating wound or not - the lung is collapsed down onto the hilum, and its excursions if they have not ceased are at least much modified in extent.

HISTORY OF PNEUMOTHORAX.

The first sign of pneumothorax which we find on record is the splashing sound produced by succussion, mentioned in the Complete Works of Hippocrates, 5th century B.C. This sign is often referred to as "Hippocratic Succussion". This sign is mentioned in the above works several times. It was supposed to be a sure sign of empyaema. In Vol. III "The Diseases", after giving the picture of a very ill person the author says, "Seat him on a seat that will not stir. Let someone hold him by the arms while you shake him by the shoulders and listen to hear on which side the sound is produced".

In Vol. V "Coacae praemotiones". "These empyema cases who, when shaken by the shoulders, produce much sound, have less pus in them than those who produce little sound." Laennec remarks that the writer of this sentence almost⁽¹⁾ discovered pneumothorax.

Celsus reports cases as empyaemata but as C, P. Emerson states in his dissertation on pneumothorax, from which the above paragraph is quoted, empyaemata seldom follow fractured ribs, and the physical signs of empyaema and pneumothorax are somewhat similar, it is possible that some of Celsus' cases may have been pneumothorax.

(1) C.P. Emerson. John Hopkins. Hosp. Reports. 1903.

One of the earliest cases in which gas was noticed to be in the pleural cavity is noted by Riolan, 1648, who remarks that he has several times seen paracentesis thoracis performed on patients thought to be suffering from dropsy, but from whose chest only air escaped.

Turning now to Artificial Pneumothorax, it was not apparently until the 19th century that the bringing about of a pneumothorax was thought of for therapeutic purposes.

The idea was first suggested, apparently, by an Edinburgh graduate, a physician named James Carson, who practised in Liverpool about 1820, and later, 1821, performed some animal experiments on this subject, predicting a great future in this form of treatment in his "Essays, Physiological and Practical".

In the year 1823, Davy made some experiments on the subject of pneumothorax, more especially as to the rate of absorption of various gases from the pleural cavity - an important subject in the choice of gas to be used in the performance of Artificial Pneumothorax. By injecting a mixture of oxygen and nitrogen, and one of nitrogen and carbon dioxide into the pleural cavity of a dog, and after so many hours killing the animal and measuring and analysing the gas remaining in the pleural cavity he determined that oxygen was absorbed faster than carbon dioxide and this than nitrogen.

In 1882 Farlamini suggested that pneumothorax should be tried as a therapeutic measure with some patients, but it was not until 1894 that he actually put his suggestion into practice, so that the credit of initiating the treatment remains with Dr. Cayley as is stated subsequently.

Pneumothorax as a therapeutic measure of sorts seems first to have been attempted in 1885 when I find that "Roser for fourteen years produced purposely on his empyaema cases, a pneumothorax, and during the last twenty years resected ribs in all cases that the opening into the pleura might be a larger one". (Deut. Med. Wochensch., Feb. 19, 1895).

But the credit of initiating the treatment of Artificial Pneumothorax as a definite therapeutic measure belongs to Dr. Cayley of London, who in the February of 1895 deliberately brought about a pneumothorax on a patient as a treatment for haemoptysis in a case of pulmonary T.B. The patient was a young man of 21, admitted to hospital suffering from haemoptysis. The haemoptysis continued practically daily and failed absolutely to respond to the treatment then in vogue, namely ice to the side of the chest, gallic acid internally, ergotin citrate hypodermically, and occasionally small, according to the present day conception - hypodermics of morphia gr. 1/6. A month later his condition is described as one of great prostration, the pulse being small and feeble and the patient blanched, having brought up more than a

gallon of blood since admission. Quoting Dr. Cayley,
 "It appeared to me that if the left lung could be collapsed, the consequent great diminution of circulation through it would afford a fair prospect of arresting the haemorrhage. It would moreover probably check at any rate for a time the developement of tubercle in the lungs. The extreme anaemia of the patient would, I thought, render this proceeding less dangerous, as less oxygen would be required for the aeration of the blood, and he would therefore be better able to support the collapse of the lung."

"Accordingly chloroform was administered, and Mr. Hulke made an incision over the 6th intercostal space, an inch behind the axillary line, and opened the pleural cavity, and introduced a double tube made by uniting two pieces of elastic catheter about three inches long by means of a shield. The opening was protected by means of a small cage lined with gauze, and the whole covered by a carbolised gauze bandage. The operation was performed with all the usual antiseptic precautions. The air passed freely in and out the tube, and the apex of the heart became displaced to the mid-sternal region. The respirations became accelerated."

The end of the case was that the patient died five days later, the haemoptysis having recurred once, then ceased. At the postmortem examination, Miliary Tuberculosis was found, and adhesions preventing full collapse of the lung.

The communication between a cavity in the left lung and a branch of the pulmonary artery was apparently shut off by clot, and the lung tissue collapsed round about it.

In conclusion, Dr. Cayley states, "As the case turned out to be one of Acute Miliary T.B., it is evident that no treatment could have prevented a fatal termination and the patient did not live long enough to enable any positive conclusions to be drawn as to the effect of the operation on haemoptysis When we consider how largely the pulmonary circulation is influenced by the respiratory process and the small amount of blood which circulates through a collapsed lung, I think such a mode of treatment offers a fair prospect of arresting otherwise uncontrollable haemorrhage."

In 1888, Potain advises that when a spontaneous pneumothorax occurs in tuberculous disease, the liquid exudate should be removed, and air which is non-irritant to the pleura, rendered sterile by filtering through cottonwool, should be substituted in its stead, remarking that the rest which the diseased lung enjoys seems to favour cicatrisation and healing of the tuberculous tissues.

HISTORY OF PNEUMOTHORAX.

In the year 1890, considerable research was done on the subject of artificial pneumothorax, in the Loomis Laboratory of the University of the City of New York. The effect of double pneumothorax on dogs was ascertained, and it was discovered that if the openings (by which the pneumothorax was made) be closed on both sides, the dog was little disturbed, while with open pneumothorax on one side and closed on the other, there was little discomfort. Bilateral open pneumothorax caused urgent dyspnoea.

In 1891 Gilbert and Roger, experimenting with dogs, made notes on the conditions of the respiratory movements and blood pressure during the performance of artificial pneumothorax. At the moment the condition is produced, they found the respiratory rhythm much disturbed. The blood pressure is little changed, but in some cases there is a drop in pressure, followed by larger slower beats, but soon regaining the normal rhythm. - *Revue de Médecine* 1891.

In the year 1891, work of an experimental character was done by Szupak, who found by experiment on dogs, that air introduced into the pleural cavity under aseptic conditions was gradually absorbed, but at the same time more than half

of pure nitrogen is unabsorbed. Moreover, he puts aside the view, formerly held to some extent, that air injected into the pleura causing inflammation of the pleural surfaces.

Meunier in 1895 did work on the same subject, and gauging the amount of gas left in the pleural cavity by the intrapleural pressure, he found that the time necessary for the complete absorption of 80-90 c.c. of air from the pleural cavity was 6 - 7 days.

Rodet and Nicholas in 1896 performed artificial pneumothorax on dogs for experimental purposes, using a very fine glass cannula to inject the air. By introducing varying proportions of oxygen and carbon dioxide in air, he determined that air introduced into the pleural cavity in dogs is gradually absorbed, at first rapidly, then slowly.

In the Lancet of 1897, West reports four cases of Spontaneous Pneumothorax in Pulmonary T.B., all of which were followed by complete recovery, but in the British Medical Journal of the same year, vigorously combats the idea that collapse of the lung favours the healing of Ruberculosis.

Hnatek in 1898 again reviews the subject of rise in Blood Pressure during the performance of ^{Bilateral} Artificial Pneumothorax, and finds a rise in fourteen cases. This he considers partly due to the stimulation of the sensory nerves of the skin etc. during the introduction of the sharp cannula through

chest wall. This rise is temporary - the permanent rise being due to lack of oxygen and dyspnoea, and the entrance of air into the pleura - this last cause being shown to be active in curarised animals in which the influence of both the respiratory movements and lack of oxygen were included.

In 1895 or about that time, Murphy reports five cases treated by the induction of Artificial Pneumothorax, but the treatment was comparatively little used until more recent times.

Aron, in 1902, did a long and detailed series of animal experiments, and concludes that when pneumothorax is produced, the respiratory mechanism must change, since one lung is rendered entirely functionless. Compensation might be obtained by the respiration becoming more frequent - the most common way in rabbits - or deeper, or both, but that as in animals so also in man, the individual peculiarities in meeting the pathological condition must be recognised.

Although as we stated the treatment was initiated by Dr. Cayley, it was soon taken up by others including Potain in France, Farlanini in Italy, Murphy in the States, Brauer in Germany, Spengler in Switzerland, Saugman in Denmark, and in this country by Dr. Claude Lillingston.

Dr. Claude Lillingston himself suffered from Pulmonary Tuberculosis and writes that in 1909 while he was a patient in

Mesnalién Sanatorium in Norway where he had been completely invalided for two years by febrile pulmonary tuberculosis, his attention was drawn to Farlanini's pneumothorax treatment, by a friend, Dr. Holmboe, who had studied it under Professor Saugman in Denmark. He states, "Having nothing to lose, I gladly took the risk of the operation. The temperature fell to normal, and the cough and expectoration ceased altogether; and the following year I returned to my work in England, where I was surprised to find the treatment was practically unknown". In August, 1910, he, aided by Dr. A. de D. Snowden and Dr. S. Vere Pearson, induced artificial Pneumothorax at Mundsley Sanatorium on a patient whose disease was mainly one-sided. The patient made an excellent recovery and two years after the operation was well.

In December 1910, with the help of Dr. Leonard Colebrook, Dr. Lillingston induced artificial pneumothorax on a patient at St. Mary's Hospital whose left lung was involved throughout, whose temperature had failed to settle, and whose opsonic index was most unsatisfactory, and reports that after alternate compression of the two lungs, complete recovery was made and maintained. These two cases are the first of their kind reported in England, but during the years which followed many hundreds of cases came to be so treated and reported upon, and the operation has now definitely come to be valued as a

recognised method of treatment in certain cases of Pulmonary Tuberculosis.

References.

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ARTIFICIAL PNEUMOTHORAX.

Having now briefly run over some facts which are of interest in the development and history of the treatment, the practical application of the operation and the means of performing it may now be considered.

Before actually coming to describe the technique of Artificial Pneumothorax, the difference between "compressing a lung" and allowing it to collapse must be realised. The former is active, the latter passive - it may be said - for in compressing a lung air is admitted under sufficient pressure to exert mechanical force upon, and cause the lung to be collapsed, while in the latter, only a sufficiency of gas is admitted to exactly counterbalance atmospheric pressure within the lung, and so to allow it to retract in virtue of its own elasticity. Thus we find great differences in the amount of gas that is required in performing artificial pneumothorax before the lung is well compressed. These differences cannot be gauged only by the size of the individual and the girth of his chest; but depend also on the elasticity of the lungs, and it is thus, for example, that an emphysematous lung, whose elasticity is diminished requires less gas to allow it to collapse than does a normal lung. The

differences in the amount of gas is well illustrated in the following table quoted from Dr. Burrell's cases.

	Amount of air.	Pressure before.	Pressure afterwards.
1.	1200 c.c.	- 30	- $\frac{1}{2}$
2.	1200 c.c.	- 8	+ $\frac{1}{2}$
3.	1100 c.c.	- 12	- $\frac{1}{2}$
4.	1000 c.c.	- 12	0
5.	1000 c.c.	- 9	0
6.	900 c.c.	- 8	- $\frac{1}{3}$
7.	400 c.c.	- 8	+ 1
8.	50 c.c.	- 4	+ 3

In the last two cases there were extensive adhesions.

The modern view has come to be that the actual gas used for the operation is in itself of no very great importance. Nitrogen, which is slightly more slowly absorbed than air, was the gas formerly largely used, but presents no advantages over ordinary atmospheric air, filtered to render it sterile, which was used in the cases reported in this thesis. An objection has been raised to the use of oxygen gas, both owing to its expense, and also to the fact that it is absorbed more rapidly than either nitrogen and air and presents no material advantage over them. M. E. Lapham, however, reports (New York

Med. Journal, 1913) that she has used oxygen with great success in cases showing dyspnoea and cardiac distress, and she alleges that oxygen has a tonic strengthening influence, instead of the depressing one of nitrogen. Oxygen has also been used as a preliminary injection with the idea of diminishing the risk of air embolism but this will be spoken of again.

As to the actual method and apparatus for carrying out the operation two operations are or were in vogue - the open operation and the method by puncture. The latter, introduced by Farlanini, is the method now used almost exclusively, and I shall therefore proceed to describe, mentioning, however, the older methods and apparatus and their modifications.

For the introduction of air into the chest some simple portable apparatus such as can be easily transported from ward to ward should be used. Various forms of apparatus have been devised, among which may be mentioned that of Dr. W. Parry Morgan, described in the Lancet, 1914.

The apparatus used at Brompton Hospital, and by which Artificial Pneumothorax was induced in the cases mentioned in this thesis, is the one, with slight modifications, which was devised by Dr. Claude Lillingston and Dr. Vere Pearson.

"It consists of a needle connected by rubber tubing to a bottle containing air, the 'gas bottle', and by means of a crosspiece with a manometer, whereby the pleural pressure is

measured. The gas bottle itself is connected with a 'pressure bottle' containing a coloured antiseptic solution such as 1 in 1000 perchloride, by the lowering or raising of which the outflow of gas into the pleural cavity can be regulated. Between the needle and the crosspiece connecting with the manometer, a piece of glass tubing containing sterilised cottonwool for filtering the air, is inserted, and a second between the crosspiece and the glass bottle. It is well also to insert a short length of glass tubing not far from the needle, so that when the instrument is used for gas replacement, the entry of fluid into the tubing may at once be detected should this occur."

This method just described was introduced by Professor Farlanini of Pavia, and was originally performed by introducing a hollow needle the size of a darning-needle, connected with a manometer, into the pleural cavity. There is a danger with this method of sharp needle of producing instantly fatal air embolism, and it is for this reason that Saugman, in the first place, fitted the needle with a blunt stilette, and later, Clive Rivière perfected this needle as described in a subsequent paragraph. The open method was up till 1913, at least, still practised successfully by Professor Brauer in Germany. It consisted in dissecting down to the parietal pleura, through an incision made over an intercostal space.

A blunt cannula, connected with a manometer and a cylinder of gas, is thrust through the parietal pleura, and if no adhesions exist at this point, several hundred cubic centimetres of gas are admitted. Subsequently, the puncture method is followed.

This open method or rather modifications of it have been tried recently. In the case of a patient where the pulmonary disease is practically entirely unilateral, but where such dense adhesions are present locally - to prevent efficient collapse of the lung - the open method may be tried as a desperate measure, where the case is obviously going down hill, and the usual treatment has not brought about improvement. In such a case, definite evidence must first be obtained by skiagraphy after possibly injecting a small quantity of air into the pleural cavity, that the adhesions are fairly local - as widespread adhesions would be of course impossible to divide entirely by surgical means. The great risk of the treatment in relation to the only problematical improvement which it may bring about, must be explained to the patient or his friends. An attempt may then be made under general, as a rule, or possibly local anaesthesia, to resect one or two ribs near the area of adhesions and separate with the fingers, and divide with instruments the adhesions and allow the lung to collapse - the so-called surgical pneumothorax. A case mentioned in this thesis was fatal

from haemorrhage, and I have also seen a case succumb to shock closely following the operation, the condition seeming to be greatly aggravated by the effects of the anaesthetic.

The relative advantages and disadvantages of the so-called "open and puncture" operations are these. While (1) the open operation does away with the danger of air embolised, and (2) lessens the danger of wounding visceral pleura and lung, the operation of incision is a more formidable operation, requiring usually a general anaesthetic which is as a rule contraindicated in these cases, there is more risk of accidental infection from without, and a fistula has been known to form at the site of incision.

Before using the apparatus, it is necessary that the tubing, filters and needle be absolutely dry, otherwise the manometer will not record the changes of pressure at the needle's point, an essential that the operation may be successfully and safely carried out. The needle should be sterilised in absolute alcohol, and any drops of water which may be present in the tubing carefully squeezed out, and the tubing whirled in the air with the object of drying it. The water in the two limbs of the manometer is brought to the same level at the point 0 before operating, by opening each limb freely to atmospheric pressure.

The preparation of the patient for the operation is not

very formidable, as a general anaesthetic is not going to be required. Drastic purgation and starvation should in all cases be avoided, but the patient should have no heavy meal within some hours of the operation, and should be kept for a day on light diet, after the first induction. An injection of gr. $\frac{1}{4}$ of morphia or $\frac{1}{3}$ of omnopon should always be given half an hour before the first induction at least, with the idea of soothing and allaying the patient's fears, and with the object of minimising pleural shock and the manifestations of the pleural reflex, of which mention will be made during a subsequent paragraph. With this same latter intention, the thoracic wall over the site of puncture must be carefully anaesthetised right down to the pleura with 2% solution of novocain in sterile saline.

The skin of the patient is prepared as for any other surgical operation, shaving being performed if necessary, and the area painted with a solution of iodine or picric in alcohol.

For the initial induction at any rate the patient should be lying down, on the side opposite to that on which the pneumothorax is going to be induced. It is essential that the patient should be comfortable. If necessary, the arm on that side may be raised above the head, and a pillow placed under the sound side, to open out the ribs, and so render the passage of the needle into the pleural cavity

more simple. Before the needle is introduced, the tube leading to the bottle of air is clamped, while the needle is left in free communication with the manometer.

The site chosen for the puncture is usually one of the axillary lines, such as the mid-axillary in the sixth space, as here the thoracic wall is thinnest, and the intercostal spaces widest. If however adhesions prevent the introduction of the needle, some other site will have to be chosen such as the eighth or ninth space below the angle of the scapula or other points at the back or front of the thorax, at the same time trying to choose a portion of pleura, as far distant from lung disease such as cavity and pleural adhesions, as possible. Should the first second or third intercostal space require to be used, great care must be taken to avoid a blood-vessel or cavity. If three or four punctures are unsuccessful, it is usually necessary to abandon the treatment, although the fifth puncture proved the successful one in a case mentioned below. Out of Professor Saugman's series of 138 cases, 43 had to be abandoned for this reason.

The choice of needle is of the first importance. Originally, as stated previously, a pointed needle was used, but the danger of wounding the lung, with the consequent possibility of producing gaseous embolism, caused this to be abandoned. The instrument described by Dr. Clive Rivière

is probably best for the initial operation. This consists of a trochar and cannula, the latter of 1.8 millimetres gauge, with a side opening near the tip and a sharpened circular end. The trochar and cannula are introduced vertically through the chosen site of puncture - some operators thrust the needle obliquely into the chest, so that when the needle has pierced the parietal pleura, it may glide over the visceral pleura without wounding it. This however increases the difficulty of gauging the depth to which the needle has penetrated - to the depth of about $\frac{3}{4}$ cm., when the shoulder of the cannula will have passed through the skin. The trochar is then withdrawn and the tap of the needle closed. The sharpened cannula is then thrust onwards, and by keeping a light hand on the needle, the slight resistance of the parietal pleura is felt, and a sudden slight jerk experienced as the sharpened circular end perforates the parietal pleura. Further confirmation should also immediately be obtained by the appearance in the manometer of a negative pressure of some 10 c.m., and definite respiratory oscillations of some 4 - 8 c.m. Until these oscillations are obtained no gas should be admitted to the pleural cavity. It may be that the needle is choked with blood or connective tissue, in which case a thrust stilette may be tried to clear it. If this fails, it is better to remove the needle, and reintroduce a fresh one, avoiding the site of the primary puncture, which

will in all probability be suffused with altered blood. Some doctors consider that if oscillations are still not obtainable, a little gas, e.g. 10 c.c., may be cautiously admitted with the idea of separating the pleural surfaces, but the possibility of the needle-point being in a vein must always first be excluded, by opening the tap, and aspirating with a syringe, the nozzle of which fits tightly into the base of the needle. If blood is withdrawn the needle must at once be removed - if not the operation may be proceeded with. Some operators, fearing air embolism, introduce oxygen only at the first induction, but Sir Percival H. S. Hartley considers this idea based on a misconception, and the practice unnecessary, provided that the needle is proved to be clear in the pleural cavity by the presence of oscillations of the water in the manometer. On the first occasion about 400 c.c. of air are usually sufficient, but this will vary with the condition of the lung and the factors previously mentioned. At the end of the first induction the intra-pleural pressure should be still slightly negative, except however when the operation is performed for haemoptysis, when 1000 c.c. of air should be introduced straightway. The sensations of the patient must also be a guide to the amount of gas introduced, extreme feeling of "tightness in the chest" - a slight degree of this is almost invariably complained of - extreme dyspnoea and cyanosis, severe fit of

coughing, the pulse becoming feeble and rapid, and other signs of pleural shock, being indications to stop. Restoratives - e.g. hypodermics of camphor, and oxygen should always be at hand - and a glass of brandy-and-water to sip at intervals during the operation, is often of moral, if not of other support to the patient.

On withdrawing the needle, the puncture should be closed with collodion and a pad of sterile gauze, a bandage usually being unnecessary. The patient, after the first induction, should be kept for a day or two on "absolute rest", if not already on it, as a means of combatting a swinging temperature, and other signs of auto-intoxication. By "absolute rest", as it is meant at Brompton Hospital, the patient is not only confined to bed, lying down, but is not allowed to perform even the slightest service for himself, such as feeding himself, nor is he allowed to read, etc.

The air for the first few days is somewhat quickly absorbed, and refills are necessary at one or two day intervals at first, later at intervals of a week, then a fortnight, and at last when the pneumothorax is well established, at intervals of four to eight weeks. The amount of air at each refill is gradually increased, until when the lung is completely collapsed; at the fourth or fifth refill it should measure $+5 \rightarrow +6$ c.c. of water. The refills should at all times be controlled by

radiographic examination - the object of the treatment being to obtain, without undue stretching of the mediasternum and discomfort of the patient, as complete a collapse of the lung as possible. The enclosed X-rays print shows the appearance aimed at, when complete artificial pneumothorax has been secured - the lung lying close against the vertebral column, and giving a definite edged shadow, like that of a solid organ, against the clear translucency of the gas which fills the pleural cavity. The patient, therefore, should be sent for screen examination after the first few days, travelling to the X-ray room if necessary as a stretcher case, and the dates of subsequent refills be timed by the skigram appearances, the dates being approximated when the lung shows a tendency to re-expand, and intervals being prolonged, when the lung remains well collapsed. The X-ray will also demonstrate adhesions preventing collapse and perhaps suggest surgical treatment. The subsequent refills may be performed with the needle described above, or Professor Saugman's sharp needle may be used, the danger of air embolism being now past.

The treatment must be continued until complete cicatrization of the tuberculous foci has occurred, unless it has to be abandoned for one or other of the reasons below mentioned. The duration of the treatment will therefore vary in individual cases - but probably in most cases should be continued for one or two years. As the general and local condition of the

** I regret I have been unable to obtain the X-ray prints which I intended enclosing. A.B.*

patient improves he will be able to leave hospital, and possibly continue his treatment at some sanatorium. Many cases in the later stages of the treatment attend Brompton Hospital as out patients; or are admitted for a night or two for their monthly or two-monthly refills, and some are able to restart and attend to their previous occupation, so little are they inconvenienced by the collapse of one lung. When the disease is chronic and has destroyed most of the lung, little can be gained by restoring the remaining small quantity of lung to functional activity; whereas there is an appreciable risk in such a case of a recrudescence, if the lung is allowed to re-expand too soon.

Considering how little many of these patients are inconvenienced by the treatment, there is no hardship in prolonging it over a considerable time, and although cases of severe but recent disease have been reported to be successfully treated by compression for six or seven months, probably the risks entailed by such a short compression are not worth taking.

Duration of the treatment must be influenced by a knowledge of the state of the disease before compression, and the symptoms of the patient before refills, more especially when the intervals between refills, short of allowing actual absorption of the gas, have been prolonged. . Occasionally,

thick-walled cavities permit of only the compression of half the lung. The disease may be arrested, but the process of cicatrization is necessarily slower, and the treatment must therefore be prolonged.

The operation is on the whole a safe and easy one to perform, but it must not be imagined for a minute that it is entirely free from difficulties and dangers. These may be divided for the sake of description into difficulties and dangers occurring at the time of operation, immediately afterwards, and what may be termed more remote complications and sequelae.

Most of the difficulties attending the actual operation have already been mentioned. These include cases where there is an absence of manometric response, due to adhesions or other reasons, such as blocking of the needle with blood and connective tissue, etc., and of these no more need be said. In some cases, where adhesions, but not dense, are present, an incomplete manometric response may be obtained, the reading recording a slight negative pressure, with feeble and irregular oscillations. In these cases Dr. Rivière recommends removing the needle, and re-entering it in another situation. In these cases of slighter adhesions, also, after apparent perforation of the parietal pleura, a large negative fluctuation - e.g. ten - may be registered, and respiratory oscillations absent. This may be either due to

blocking of the needle, or of entry of the needle among loose pleural adhesions where the manometer gas is lost: occasionally on the perforation of the pleura, the usual manometric variations occur, but on admitting only 60 - 70 c.c. of air, it is found that the intrapleural pressure is now greater than the atmospheric, and only weak respiratory fluctuations are recorded. It is probable in this case that the needle has entered a pleural pocket. Gas may be admitted up to a pressure of say + 5 c.m. of H_2O , and if there is any outlet, the pressure will gradually fall, as the gas escapes into the general pleural cavity. If this does not occur, gas may be admitted cautiously up to + 10 or + 15, in an attempt to force an opening into the general peritoneal cavity, but no more at the first operation; should this fail, the needle should be removed and re-entered.

The chief dangers of the operation, apart from those of any operation of paraventesis thoracis, such as wounding of an intercostal vessel, etc., which can be avoided by ordinary care, are probably "pleural shock", cases of gas embolism, and wounding of the lung; and of the first two mention has been made in passing. Rarer dangers, such as puncture of the abdominal cavity through an unusually high diaphragm - and recognised by a negative pressure, with reversed respiratory fluctuations, have been mentioned.

The symptoms of pleural shock are usually indistinguishable from those of gas embolism. The symptoms come on as a rule with great suddenness, usually at the moment the needle is entering or leaving the pleural cavity. Less commonly they appear after an interval of twenty minutes or so, or even after some hours. The typical case presents sudden pallor with loss of consciousness, soon to be followed by cyanosis, and irregularity of pulse and respiration. Dilatation of the pupils occurs, and clonic and tonic spasms of the limbs, occasionally only on the side of the pleural reflex. There may be an initial cry. Farlanini divides the symptoms into the three groups of psychic, motor and circulatory. Of course, as Dr. Rivière points out some of these symptoms may be in reality of embolic origin. De Cereville considers that a flaccid paralysis points to embolism.

It is a peculiarity of pleural shock that it tends to occur at refills rather than at the initial operation, and once having occurred, tends to recur and increase in gravity at subsequent operations, and for this reason Farlanini considers that after its second occurrence, the treatment in that particular case should be given up. The condition must only be diagnosed as "pleural shock" when gas embolism has been excluded. Sachs reports twenty-six cases of pleural shock, none fatal, out of 1058 cases of pneumothorax

in American literature. Some observers - De Carle Woodcock and J. A. M. Clark - consider that pleural shock is more frequently met with in cases of early disease with healthy pleura - and this may be considered against the treatment of early cases of phthisis by Artificial Pneumothorax.

Zesas (Zentralblatt f. Chirurgie, 1914) has been able to collect from literature 54 cases of serious pleural shock. In animals shock can be caused by an injection of a chemical irritant such as tincture of iodine into the pleural cavity, but not by mere mechanical irritation. Cordier found that shock occurred mainly through the vagus nerve, and could be avoided by a preliminary injection of morphia, as recommended previously, and careful anaethetising of the thoracic wall, including the parietal pleura with novocain. In spite of these precautions, should it occur, the needle should be withdrawn, the patient's head lowered, and a hypodermic of some stimulant, e.g. camphor, which should be at hand, as suggested, should be administered. Saugman recommends faradisation of the heart and phrenics, and as a desperate measure the intra-venous injection of digalen.

Gas embolism is symptomatically and even post mortem difficult to distinguish from pleural shock. The recurrence of the symptoms at more than one refill points to pleural shock, while Brauer has stated that the discovery of gas bubbles in the retinal vessels is definite proof of gas

embolism. Gas embolism is as a rule brought about by puncture of the lung or the wounding of a vein, which has developed angiomatous proportions in a pleural adhesion. A case has also been recorded where it followed on puncture of a vein in the thoracic wall. These dangers may be obviated by the measures described previously, that a blunt instrument should be used, that no gas be injected until respiratory oscillations appear, and a negative pressure is recorded - this latter will not suffice when it is remembered that the pressure with the veins may be negative - and that oxygen, which is a gas more soluble in blood, especially venous blood, be used at the initial operation. The treatment is fortunately very similar to that indicated for cases of pleural shock, but artificial respiration which may be used in the latter, must be strictly avoided in the former, where there is danger that more gas may be pumped into the vessels.

Dr. Clive Rivière, whose views are largely recorded in the above paragraphs, considers that the most serious accident to which a pneumothorax patient is liable is perforation of the lung. The condition is of great rarity - Farlanini has 8 perforations in a series of 139 patients. The condition appears predisposed to, when large, especially superficial, cavities are present, and when the presence of

adhesions prevents complete collapse, and the patient attempts to live his ordinary life. Marshak and Craighead (American Review of Tuberculosis, 1917-18) report a case of Spontaneous Pneumothorax during a course of treatment by Artificial Pneumothorax. The symptoms are sudden severe pain in the side, followed by a hectic temperature, and the rapid appearance of a pleural effusion. The condition is usually followed by a fistulous communication between lung and pleura, with continual reinfection of the pleura, and death from empyaema within a year. Spengler (Münich Med. Wochung, 1913), devised a method of attempting to cure such cases, which are incurable by the ordinary methods of empyaema treatment, as the lung cannot expand and obliterate the cavity, by a series of aspirations each followed by a plastic operation, and reports seven cures out of thirteen cases so treated. Among the prophylactic measures may be mentioned the limitation of activity in patients with partial pneumothorax, and the treatment of patients by Artificial Pneumothorax, prior to the advancement of the disease to the stage of large cavity formation. Stivelman also includes in the immediate dangers injury to heart muscle or puncture of ventricle - he records a case of the former - and cocaine poisoning. He has known .08 gm. prove fatal where idiosyncrasy exists.

Of the dangers occurring just after or during the performance of the operation, little apart from what has been mentioned above need be said. The occurrence of a slight degree of surgical emphysema around the site of thoracic puncture occasionally occurs, as is noted in some of the cases in this series, and rarely causes any inconvenience. The spread of air is best prevented by the application of a pad and bandage, and the prevention of unnecessary coughing. Occasionally gas passes under the visceral pleura into the interstitial lung tissue, where it may track up the mediasternum and appear under the cervical fascia in the night, causing a feeling of tightness - perhaps dysphagia and cyanosis. No treatment is usually required.

The severe dyspnoea which occasionally occurs after inducing pneumothorax is best relieved by inhalations of oxygen.

Occasionally, when pneumothorax is done more or less as a desperate measure, the treatment may have to be rapidly abandoned. This happened in a case which was under my care at Brompton Hospital. It was one of a young girl of about twenty years of age, who had signs of disease, which was progressing on both sides of the chest, but slightly more marked on one side than on the other. In spite of the patient's having had previous treatment, and being kept for

several weeks on "absolute rest" (for explanation, see before), the temperature showed absolutely no signs of settling, the disease was progressing, and the patient's mental and physical condition deteriorating daily. The case was obviously one for desperate measures, and the risk having been explained to the girl and her mother, it was decided to attempt to compress the more affected lung. After the original induction and first refill, the patient, in spite of being very dyspnoeic, seemed a little better, and the temperature even showed signs of settling. Then the dysonoea became rapidly worse, the respirations increased in rate and the temperature rose, and the girl was found to have developed a pneumonia on the side of the one active lung - probably of tuberculous nature, so-called acute tuberculous pneumonia. The question of decompressing the collapsed lung arose, by perforating the thoracic with a hollow needle, and allowing air to escape, but it was not thought worth while, as the treatment had only been instituted a few days, and the gas would be rapidly absorbed. Apparently, the one active lung being also diseased, had, when it was called upon to perform the work of both lungs - that compensation does come in is shown by the presence of harsh prolonged breath sounds over the uncompressed lung - been unequal to the effort, which also brought about an acute

exacerbation of the disease - which later proved to be fatal.

Of the sequelae the only one that need be mentioned is that of pleural effusion.

Rivière puts this complication or sequela down as happening in about 50% of cases kept under observation from start to finish, and this he considers due to the mechanical factors present in the condition of pneumothorax. It is well recognised that pleurisy with effusion occurs at the initial onset of pulmonary tuberculosis with considerable frequency, but it is not of nearly so frequent occurrence, in cases in which the disease is well-established in the lungs, and infection of the pleura by small surface foci of T.B. shut off by the formation of adhesions, when, however, by the artificial formation of a pneumothorax, the pleural surfaces are separated and the diseased lung collapses, any small focus of T.B. present on the pleural surface can readily infect the pleural cavity and set up a pleurisy, with or without effusion. It is for this reason that the effusions are found generally to be sterile on culture, but to contain tubercle bacilli either to microscopic examination or to animal inoculation.

The occurrence of ordinary dry pleurisy may be ushered in by pain in the side, etc., but if the pleural surfaces are well separated by gas, pain is not usually severe, and

the chief symptoms are then some febrile reaction and vomiting. A localised pleurisy may occasionally appear at the site of puncture.

This may be followed by a mild serous effusion, small in amount, and tending to disappear.

But large pleural effusions may occur, reaccumulate several times after removal, and remain in the chest for long periods. In these cases the typical signs of hydro-pneumothorax may be demonstrated, including "Hippocratic Succession". These effusions frequently remain serous, but may either at once become purulent gradually with the formation of a pyopneumothorax, which may be of so-called "benign nature" - a tuberculous pyopneumothorax, or of serious type caused by infection by pyogenic organisms from the outset.

Mayn has proved by experimental work (Beiträge z. Klin. der Tuberk., 1914), that the serous effusions may be regarded as inflammatory in nature, their specific gravity coming above 1018, the figure accepted as the lowest limit for inflammatory effusions. The cellular content is as a rule low, except in mixed infections. The bacteriology has already been mentioned, and the universality of the T.B. Bacillus is shown in the fact that in Saugman's series of 138 cases, he only failed to find T.B. bacilli twice in all his cases of large effusion.

The effects on the pneumothorax treatment may be

favourable or unfavourable. As a purely mechanical agent, the effusion of fluid may assist in the breaking down of adhesions, and so allow the pneumothorax to become more complete; but apart from this, some benefit may result from the serological effect of the effusion. The gas also is not so rapidly absorbed from the pleural cavity, so enabling longer intervals to be left between the refills. Among the unfavourable effects, apart from those which will of course be present with a purulent effusion, is the fact that the effusion may cause such marked thickening of the visceral pleura that when the Artificial Pneumothorax treatment is over, the collapsed lung may fail to re-expand.

With the occurrence of symptoms suggesting the onset of a dry pleurisy, etc., the patient should be confined to bed, and have warmth applied to the affected side. Saugman considers that in these cases salicylates exert a specific effect, and they should be therefore administered. Small serous effusions may be left alone, and the same applies to large chronic effusions, which are not causing inconvenience. The patients are frequently not at all disturbed, but some gas should always be kept above the fluid in the pleura, to keep the lung collapsed by an effectual positive pressure, and should dyspnoea or other signs of distress occur, the fluid should be evacuated and replaced by gas, as in the treatment

of pleural effusions by "gas replacement". For the operation of "gas replacement", in addition to the usual artificial pneumothorax apparatus, a Potain's aspirator exhausted ready for use, and with needle, is required. With the patient in a recumbent position, the Potain needle is entered at a suitable spot, and a little fluid is drawn off to establish its existence. The tap is then closed. At a point where good resonance on percussion indicates the presence of gas, the pneumothorax needle is entered, and when manometric variations establish the fact that the needle is within the pleural cavity, air is run in, as, by reopening the aspirator tap, fluid is run out. If the intra-pleural pressure is excessive a little fluid may be run off first.

When all fluid is exhausted as shown by the presence as shown by froth and air escaping through the Potain's needle, this is withdrawn, and the puncture sealed carefully with collodion. Then gas is run in, until the manometer shows an intra-pleural pressure of $+5$ or $+6$ c.c. of water. This may require to be repeated several times. Autosero-therapy, as in ordinary serous effusions, may also be tried, 1 c.c. of the exudate being injected hypodermically immediately on its removal from the pleural cavity.

The acute serous effusion may be a manifestation of an

intolerance to the increased intra-thoracic pressure, and this may be a reason for giving up the treatment. The symptoms and fever usually ameliorate, however, on the removal of the fluid and its replacement by gas. The purulent effusions must also be treated on these conservative lines, as the open operation may lead to intractable sinus formation - some authors, e.g. Farlanini and Spengler, have found that removal of the fluid and washing out the pleural cavity through a pneumothorax needle with, e.g. $\frac{1}{2}$ - 1% lysoform solution the best form of treatment.

Patients at the onset of pneumothorax treatment, especially when the left lung is the one concerned, complained of symptoms of dyspepsia, etc. Palliative measures are generally sufficient because the symptoms tend to disappear, as the thoracic and abdominal organs accommodate themselves to the changed conditions.

About the actual mode of action of pneumothorax, much discussion has taken place, but generally speaking it may be said to produce its beneficial results, by either of two ways - by its mechanical effects and by its serological effects.

The former of these was probably the one to first attract the attention of earlier workers on the subject, to whom the treatment was first suggested by the beneficial effect which occasionally follows on a case of pulmonary

tuberculosis in which spontaneous pneumothorax has occurred. These effects may be divided for the sake of description into those which follow as the result of the passive collapse of the lung, and those which follow upon active compression by the positive pressure of air within the pleural cavity. By the first of these, the lung is put at rest, and the good results which follow are said to be due to its immobilisation, as in any diseased structure, the bactericidal agents and other aids in the process of healing being given their full opportunity for action, and a process of arrest of disease, falling in of abscess cavities, healing and fibrosis set up.

Among the more active of the mechanical effects may be cited that the actual compression of the lung by the gas forces purulent secretion out of dilated bronchial tubes, and bronchiectasis. In the treatment of haemoptysis it may be said to act passively in that the collapse of the lung brings about rest and so lessens haemorrhage, which may be forced out by active movement, but also actively, the actual compression of the lung diminishing the amount of blood circulating through it and the pressure of the collapsed lung exerting itself on the lung tissue surrounding the spot from which the haemorrhage arises, and the occlusion of the bleeding point by clot.

Among less obvious methods of this mechanical action may be mentioned the effect on the T.B. Bacillus of the

diminution of oxygen in the collapsed lung, and the rapid growth of fibrous tissue - the tubercle bacillus in many of these cases disappearing from the sputum.

The swinging type of temperature, and other constitutional symptoms and signs present in some of the cases in this thesis, are obviously manifestations of an auto-intoxication, the toxin being absorbed chiefly by the lymphatics from the disease foci in the lungs into the general circulation, there to produce by their action the constitutional signs, e.g. night sweats, emaciation, etc., which we have learnt to associate with a progressive case of pulmonary T.B. By the favouring of fibrosis and obliteration of cavities, etc., the diseased source of toxin in these foci is diminished, and the diseased surface is greatly reduced in area and therefore in absorbing surface. Also some workers, Bruns, etc., have established the fact besides the lymphstasis brought about by the process of immobilization, the lymph channels are distended and absorption reduced to a minimum.

Another view to account for the beneficial action of this treatment is advanced by Molle, who attributes the success of the treatment to a "trophic excitation" of the vagus nerve ending in the lungs.

Professor Saugman considers that the treatment has a beneficial action on both lungs, stating that just as in

mild febrile cases of T.B. a certain amount of exercise is allowed, so in severe febrile cases, the patient is kept on rest. In the lungs the more diseased is kept at rest, whereas the slightly diseased is allowed exercise. That this increase of exercise may prove too much for the better lung is shown in the case previously quoted, where acute tuberculous pneumonia developed.

Although there has been diversity of opinion as to what cases are most suitable for artificial pneumothorax treatment, certain fundamentals are agreed on. Thus that among definite contra-indications to the treatment may be enumerated, advanced bilateral pulmonary disease, with extensive cavitation - especially when these have been demonstrated clinically to be superficial, serious cardiac or renal complications and constitutional disease that of itself makes recovery impossible. Tuberculous laryngitis was thought at one time to definitely contra-indicate this treatment, but since Dr. S. V. Pearson's success at Mundesley Sanatorium with a case of severe laryngeal tuberculosis combined with unilateral T.B. of the lungs, which has been followed by a similar experience in others, this is not now regarded as a definite contra-indication.

Tuberculous disease of the intestine with diarrhoea is usually against the treatment, unless mild and early, when it may undergo cure. Cases have been recorded when death took

place after apparently successful artificial pneumothorax from T.B. meningitis and T.B. Pyosalpingitis. This is in itself a plea for early pneumothorax treatment, before those complications which are frequently secondary to a primary focus in the lung have developed. An early unilateral case of Renal T.B. or T.B. of large joint that can at the same time receive surgical treatment, is not in itself a contra-indications. The treatment must in all cases be aided by dietetic and hygienic measures (Balboni, Boston Med. J., 1912).

The case which is ideally and classically suited to the treatment is one of so-called "Clinically Unilateral T.B.", by this being meant, that the disease so far as physical signs are concerned, is limited to one lung. Subsequent progress of the case on X-ray examination may reveal the presence of tubercle in the at the time clinically sound lung, but if at the time of proposed treatment the disease in the lung is not of sufficient activity to make itself revealed clinically, and if on X-ray examination, signs, fairly slight, of healed or fibrous phthisis are present, these may be ignored, and this lung treated as the better lung - always remembering that this lung will require all the more careful examination from time to time, lest the double work imposed upon it may reawaken old healed T.B. mischief.

Rivière considers that of the two, T.B. commencing at the hilum is more favourable for the treatment than apical commencing T.B., the latter being much more frequently complicated by active disease in the better lung.

Two courses are open in the matter of treatment - either the unilateral diseased lung, if of the classically suitable nature, may be compressed straightaway, see cases later. showing good results, gain in weight, etc., or as is more often done at Brompton, the effects of hygiene and treatment and rest are first tried, and if these are found ineffectual in reducing the temperature, and arresting the progress of the disease, the active lung is then compressed. This method is also adopted in bilateral disease, where one lung shows slight clinical manifest, and the other more advanced, with considerable success in some cases, as is shown in the notes.

Occasionally, cases of fairly marked bilateral disease are treated as a more or less desperate measure. See case quoted. In all cases it is the more affected lung that is compressed, in order that the better lung may be left to carry on the double work.

As a rule definitely very early cases are not treated by this means, for as Rivière points out, they are eminently curable by dietetic or hygienic measures, and they should not therefore be put even to the slight risk of pneumothorax

treatment, especially as they are more prone to show signs of pleural shock as stated before.

Cases of severe and repeated haemoptysis have been treated by this method, when they have resisted other forms of treatment, but the patient should not be allowed to come into moribund condition. Repeated small haemoptysis may be also so treated.

In cases of severe haemoptysis remarkably good results have been obtained.

Other things being equal, patients of calm, phlegmatic temperament have better chances of success than those of an excitable, anxious nature, and this should be taken into account in choosing a case for H.P. treatment.

The presence of very dense adhesions obliterating the space between lung and pleura must of necessity be a mechanical contra-indication, but in cases where apparently one or two fairly dense adhesions exist on X-ray examination, pneumothorax treatment, its results being watched and controlled by careful and repeated skiagraphy, may be attempted, if the case is an otherwise suitable one. Adhesions may be stretched or ruptured by such treatment and collapse of the lung brought about, and even if partial collapse only is brought about, much benefit may result to the patient. If after repeated attempts to induce pneumothorax, only a very small quantity

of air can be introduced, and the lung shows no sign of collapsing, the treatment will have to be given up.

Bases of Bronchiectasis are often favourably influenced and healing brought about by pneumothorax treatment, as stated before. The treatment should at all times be aided by hygienic treatment, the administration of autogenous vaccines, the exhibition of creosote internally, and any other ordinary means of treating the condition.

Summarising then, the treatment is an ideal one for cases of clinically unilateral pulmonary T.B., which is progressing, or to other cases where one lung is as yet slightly affected, but where the case has not yielded or improved under a three-months' hygienic and sanatorium treatment, provided always the general condition of the patient is fair, and the disease not too far advanced. It must be remembered that in these advanced cases of disease, although temporary benefit, as shown by the reduction of the temperature, may occur, yet the cases frequently terminate fatally in three to four months. The treatment is also a valuable one in cases of haemoptysis and bronchiectasis, and may occasionally be adopted as a desperate measure in bilateral disease resisting other treatment, always remembering the risk incurred thereby. For such cases, an attempt, without any very satisfactory result, has been made to compress the two lungs alternately.

If the treatment is successful, the temperature falls, pulse rate slows, and the general condition of the patient as regards weight, sleep and appetite improves, while the expectoration lessens in amount, and physical signs, such as crepitations, etc. disappear in the compressed lung. If the case is being treated for haemoptysis this ceases. The treatment may require to be abandoned on account of dense adhesions present preventing collapse, where pleural shock has been manifested, when the general and local findings do not improve, or deteriorate, and in cases where the "better lung" shows itself incapable of the increased strain put upon it by the development of signs of activity hitherto lateral, or the onset of acute pneumonia, pleurisy, etc.

The results obtained by various authors are on the whole satisfactory, though differing considerably, as of necessity they must, the same class of cases not being selected by all operators. Thus, Saugman, addressing his remarks to XVII International Congress of Medicine, 1913, commences his account by reminding his audience that "this treatment deals with very severely attacked third-stage patients, of whom a great part, indeed most of them, without this treatment, would have practically no chance of recovery and healing". Thirty-seven of his cases he classifies as "quite hopeless".

In the other cases, the treatment in all was commenced over nineteen months ago, before the time of speaking, and the results in these cases Saugman classifies as follows:-

	<u>Pneum. effect.</u>	<u>Pneum. failed.</u>
1. Able to do ordinary or light work.	32	8
2. Unable to work on account of T.B.	18	12
3. Dead from T.B.	12	14
4. Unknown.	0	1
5. Dead from acute compli- cations.	2	1

Not considering the last three the percentages are as follows:-

1. Able to work.	50%	22.2%
2. Unable to work.	28.1%	33.3%
3. Dead from T.B.	18.7%	38.8%
4. Freed from T.B. bacilli.	50.0%	8.6%

The difference between the two series of cases is obvious. Column 2 is what one may expect from sanatorium treatment only, while Column 1 gives the results which may be obtained in exactly the same class of patient from sanatorium treatment only.

Theodore Sachs, writing in the "Journal of the American Medical Association, Nov. 27, 1915, reports the conclusions arrived at from this treatment by twenty-four American

observers. The cases reported on are 1145 in number, of which 912 or 88.7% were in the far advanced stage; 109 or 10.6% moderately advanced; seven or .7% incipient: bilateral involvement in 691 cases or 77%, unilateral 206 or 23%. Unimproved, failed, dead - 49.1%. Improved, arrest, cure - 21.7%.

The efficacy of the treatment can only be judged by the subsequent course of the case through a long period of years, and Dr. Sachs gives 12% as the figure thus shown to be cured or arrested.

In conclusion, and quoting results nearer home, may be quoted the results given by Dr. Esther Carling - analysis of fifty-four cases so treated at Berks. and Bucks. Sanatorium. All had failed to respond to ordinary sanatorium treatment.

- (1) In 12 cases it was impossible to effect pneumothorax, and of these 10 are now dead.
- (2) In 8 temporary improvement - of these 2 dead, 6 at home unable to work.
- (3) 22 marked improvement, of which 13 have returned to their old occupation, and to a moderately active and useful life.

Considering the type of case treated, the progressive and hopeless outlook of the disease in many cases, these results may be said to be distinctly hopeful. More may be hoped for with the social and educational betterment of the

lower classes, cases which show promise of cure, often relapsing on return home, where hygienic conditions are not of the best, and hospital regulations and precautions neglected or forgotten.

CONCLUSION.

1. The treatment has now been a definitely established therapeutic measure for some fifteen years, and during that time has undergone much betterment in technique and choice of case.
2. The operation is on the whole a simple and safe one, provided that ordinary precautions are observed, and the apparatus is used by one, who knows and is accustomed to its use.
3. That thought dangers exist, these are known to be comparatively uncommon, and have largely been obviated by improved apparatus and technique and the methods of prophylaxis quoted in this thesis.
4. The treatment must ^{not}~~now~~ be considered an alternative, but rather as an adjunct to sanatorium and other treatment, and after return home, as careful attention to details of hygiene, rest, etc. are required, as in cases of P.T.B. treated by any other means.
5. That the treatment must be continued for a certain period of time to be valuable, and that although cases have

been reported as cured after seven months' treatment, two years is usually the least that can be recommended with any safety. This necessitates the attendance at the O.P. of or admission into some hospital which possesses the apparatus for this treatment from time to time, the inconvenience of this being frequently mitigated by the fact that the patient can frequently undertake his work in the intervals.

6. The treatment is suitable and can be performed in a private house or nursing home, but for the fact that it must be controlled by expert radiography, necessitating that the initial operation and first few refills, etc. should be performed in hospital.

7. The choice of suitable cases is the all-important matter, both in order that good results may be obtained and fatalities avoided, but that public opinion and that of general practitioners be obtained, and not alienated by the treatment and its results.

In the cases described in this thesis as suitable, the best results may be looked for, and a standard of hope for cure established in a disease which has often been looked on as hopeless of cure.

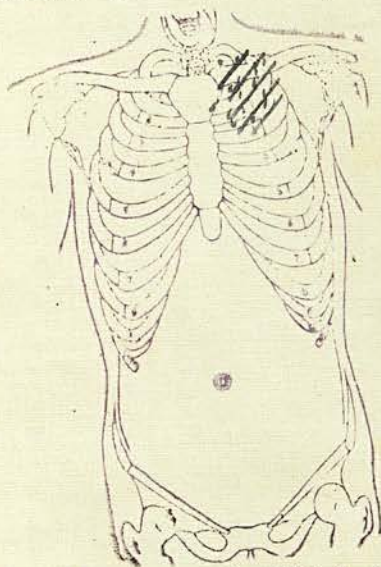
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 2. English and Latham's System, 1912.
 3. Dr. Claude Lillingston, Practitioner, 1913.
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 9. L. Hammon. John Hopkins Hospital Reports, 1913.
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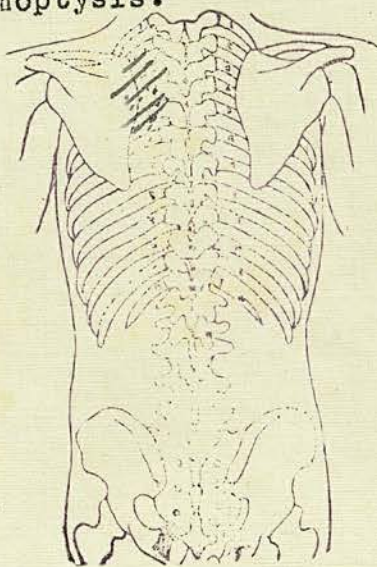


CASE 1. MICHAEL TEBBITT.

Shows gain of weight and general improvement, and cessation of haemoptysis.



Heart Nil.



Done for clinically unilateral disease & history of repeated haemoptysis

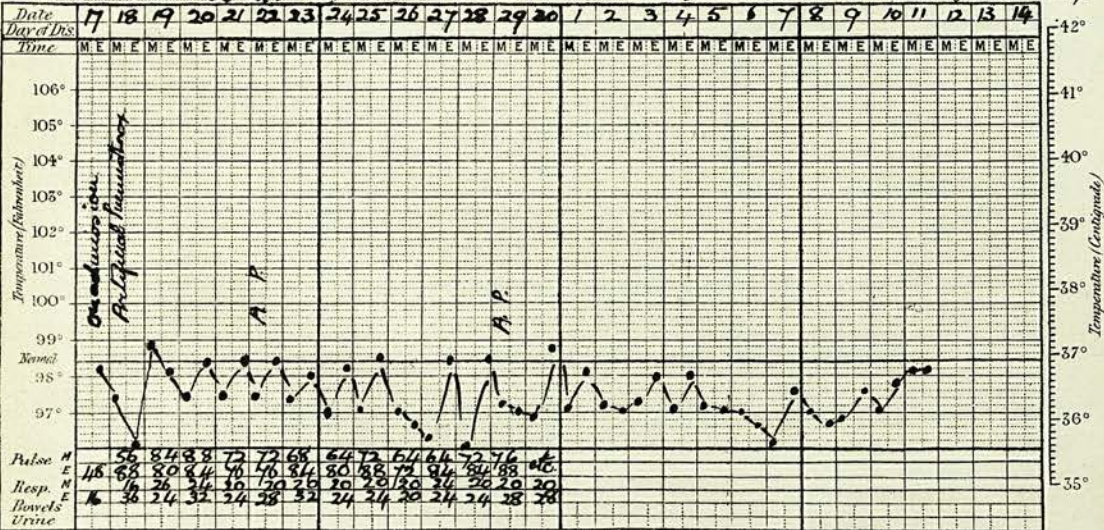
Ad 17.4.19.

Sept '19

October '19.

Gained 5 1/2 lbs.

Name Michael Tebbitt. Age 28. Disease Pulm. Tub. Result General improv.



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10, 1919.

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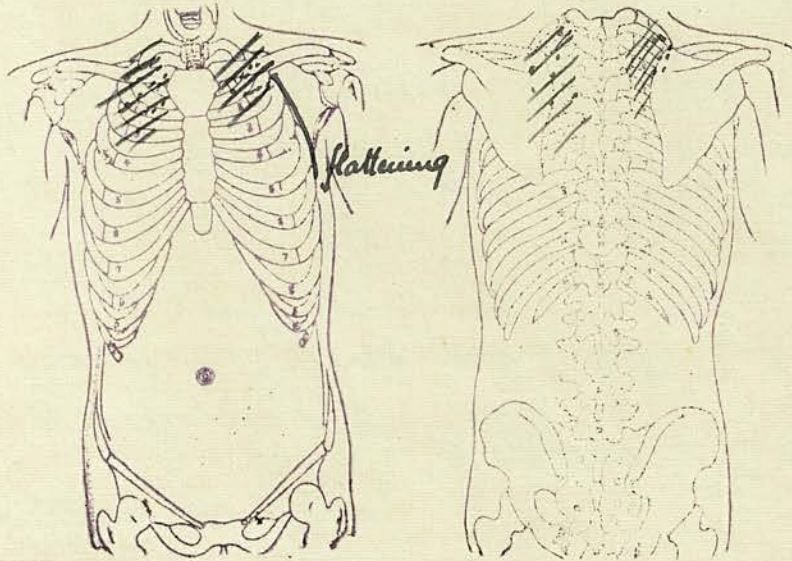
Auscult. A few crepitations present,

18.9.19. A.P. performed. 1100 c.c. Press. - 1/2

- 20.9.19. Art. P. performed. 700 c.c. Press. + 2
- 22.9.19. X-ray. Lung well collapsed.
- 22.9.19. A.P. 500 c.c. + $3\frac{1}{2}$
- 26.9.19. Left limit cardiac dullness now at rt. border of sternum.
- L. side shows little movement.
- B.S. are weak with amphoric breathing towards base. Slightly tympanitic. Bell sound in front and in axillary line.
- 29.9.19. A.P. 800 c.c. P. + 4
- 17.11.19. X-ray. L. lung decompressed. Good movement of diaphragm.
- 18.11.19. 800 c.c.
- 24.11.19. 900 c.c.
- 8.12.19. 800 c.c.
- 12.12.19. Pneumothorax complete.
- 5.1.20. Good collapse of 1 lung. Dis. Home Imp. gain $5\frac{1}{2}$ lbs.
- No evid. dis. Haem. ceased.

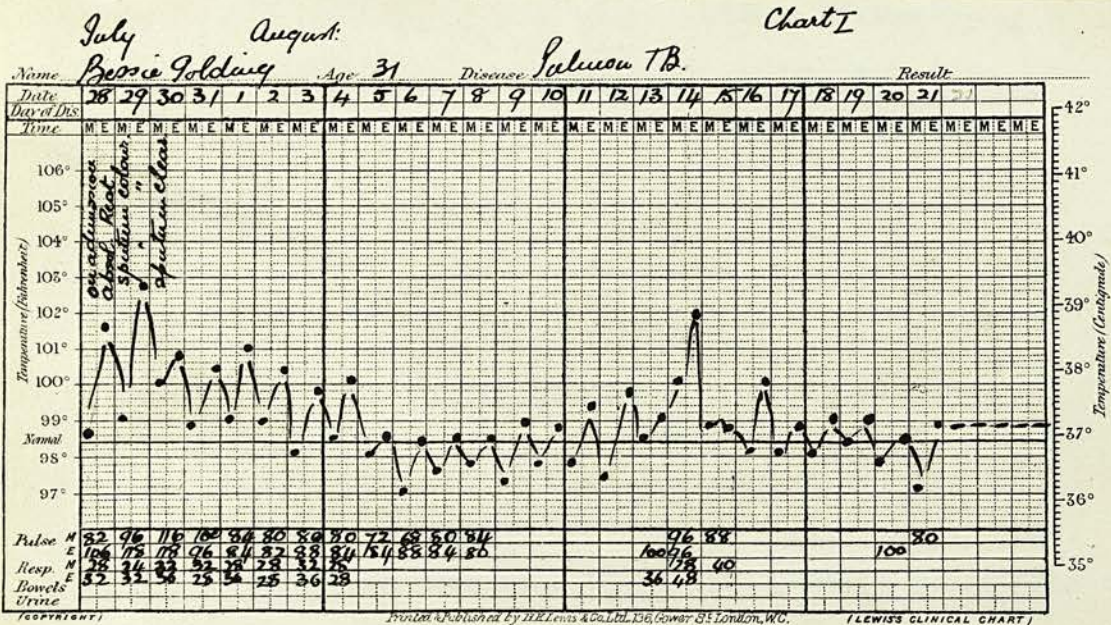
CASE 2. BESSIE GOLDING.

Inspn.
harsh.



(For continued temp., etc. and mainly leftsided disease - progressing. Followed by slight surgical emphysema.)

Bessie Golding. Aet. 31. Ad. 28.7.18.



tered.

Left apex markedly deficient + a little flattening.

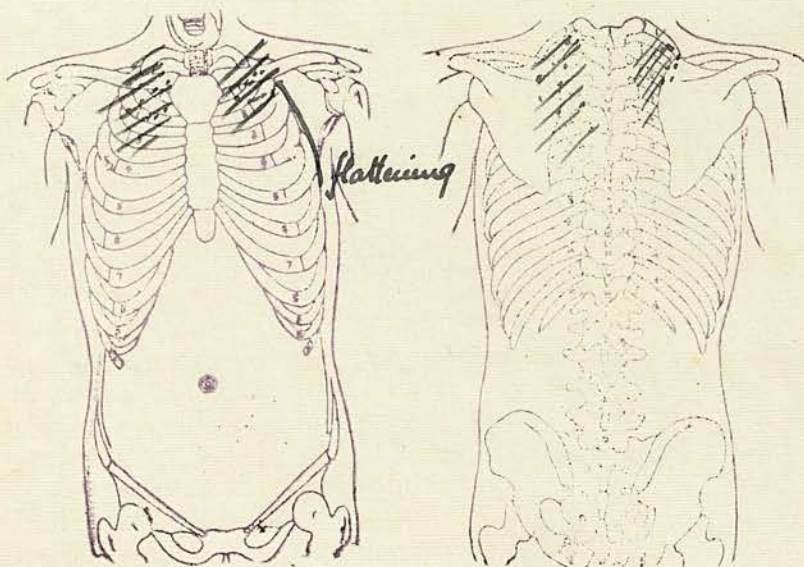
Palpat. Confirms above. V.F. a little + at left apex.

Percuss. Note impaired at both apices.

Ausc. Inspir. harsh at rt. apex.

CASE 2. BESSIE GOLDING.

Inspn.
harsh.



(For continued temp., etc. and mainly leftsided disease - progressing. Followed by slight surgical emphysema.)

Bessie Golding. Aet. 31. Ad. 28.7.18.

Dis. 17.5.19.

Diagnosis: Pulmonary T.B.

T.B. + 26.8.18.

toms, etc.

Cough +
Expectoration +
Pain in chest +
Haemoptysis +
Dyspnoea +
Night Sweats -

sical Signs on admiss. Inspect. Chest wall well covered.

Left apex markedly deficient + a little flattening.

Palpat. Confirms above. V.F. a little + at left apex.

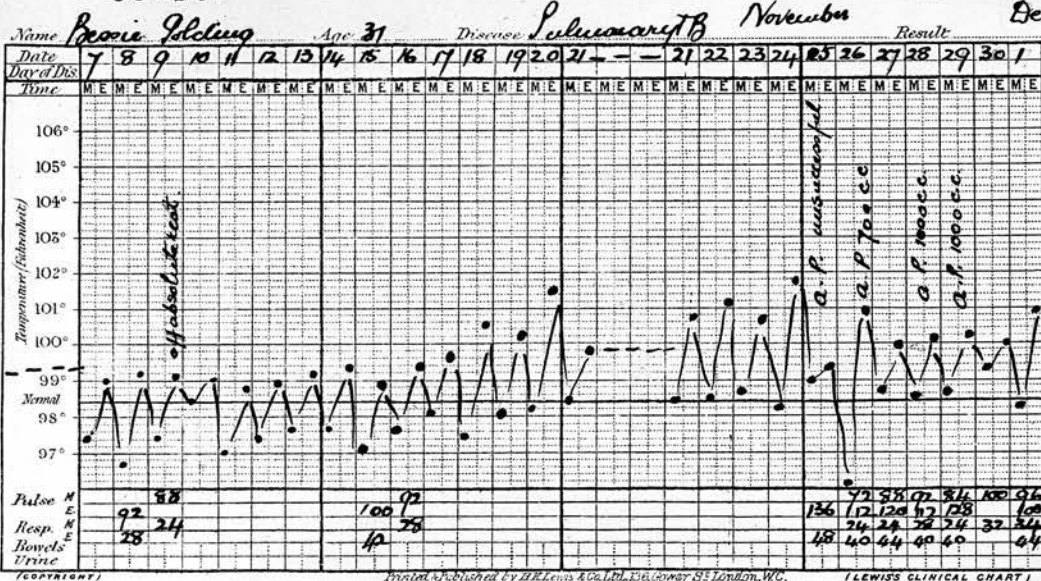
Percuss. Note impaired at both apices.

Ausc. Inspir. harsh at rt. apex.

October.

Chart II

December.



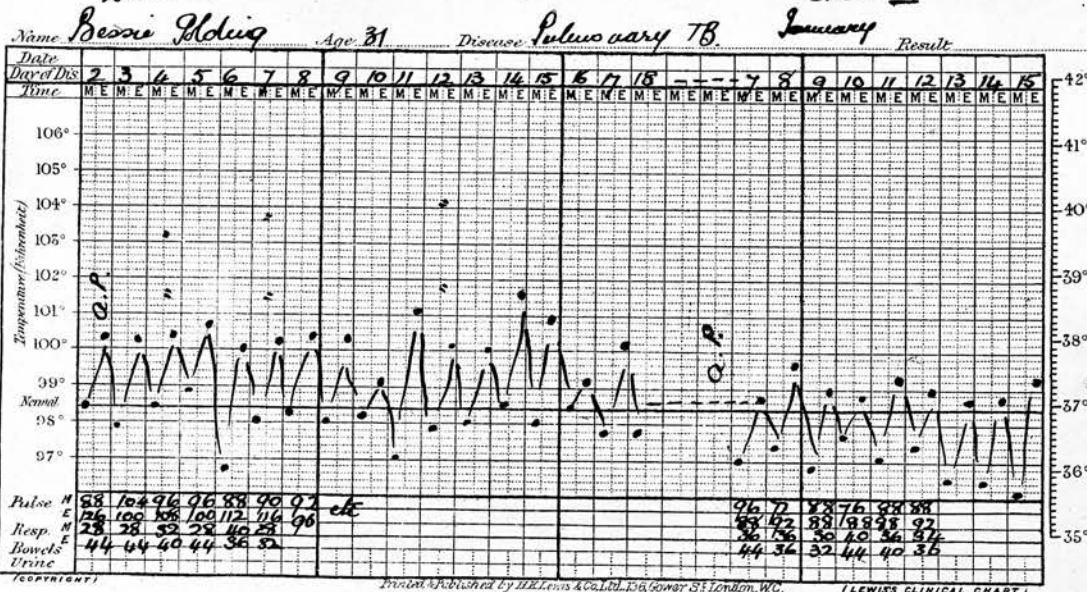
ally very troublesome.

Nov. 18. No added sounds heard over rt. lung. L. lung bubbling râles in front and behind. No sign of any arrest of disease.

Nov. 25. Three attempts were made to induce sputum without success owing to adhesions

December

Chart III

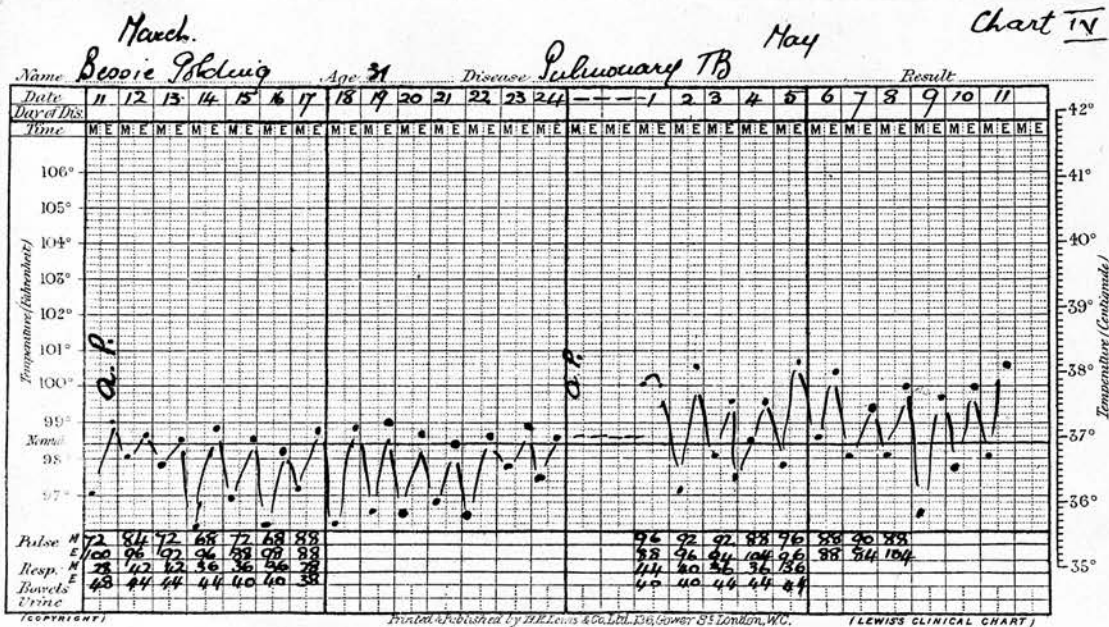


Dec. 12. 700 c.c. air.

Dec. 17. Cough still very troublesome and sputum copious. General condition was improved.

Scattered creps. at both apices.

- Aug. 26. P.S. unchanged. E. temperature usually raised.
Occasionally coloured sputum.
- Oct. 2. Temperature more satisfactory, but cough not gone.
- Nov. 6. Dullness over left lung has increased behind and postero-laterally. Cough still troublesome and sputum increased in amount. Appetite not at all good and tongue coated. Dyspnoea occasionally very troublesome.
- Nov. 18. No added sounds heard over rt. lung. L. lung bubbling râles in front and behind. No sign of any arrest of disease.
- Nov. 25. Three attempts were made to induce A.P. without success owing to adhesions between pleura. Patient much pain.
- Nov. 26. Two attempts made, second successful, 700 c.c. of O. introduced. Some surgical emphysema.
- Nov. 27. 1000 c.c. of air introduced without difficulty. Patient fairly comfortable. Some surgical emphysema.
- Nov. 29. 1000 c.c. of air easily introduced, followed by rather severe dyspnoea, which was relieved by administration of oxygen.
- Dec. 2. Press. - 4 - 2. 1000 c.c. air - 2 + 2 + slight dyspnoea.
- Dec. 6. - 2 + 4. 500 c.c. air.
- Dec. 12. 700 c.c. air.
- Dec. 17. Cough still very troublesome and sputum copious. General condition was improved.



... of persistent adhesions. These prevent complete obliteration of all the air-containing spaces in the lungs, and this is probably the reason for the persistence of large quantities of sputum.

Further pressure is not indicated as the heart and mediasternum have been pushed to the right side to the extreme limits of comfort.

Later it may be possible to keep lung in its present condition with a smaller pressure.

- 9.4.19. 400 c.c. air.
- 23.4.19. E.T. raised for last fortnight.
- 30.4.19. 200 c.c. air.
- 14.5.19. A.P. complete.

6.1.19. Cough still troublesome. T. more settled, expectoration less and general condition much better.

5.2.19. 1200 c.c. air introduced under pressure of 5 c.m. of water.

This is the most that has been introduced at one time. re advisable, as patient became very dyspnoeic.

24.2.19. 16000 c.c.

10.3.19. 14000 c.c. + 4 c.m.

Last X-ray shows a satisfactory collapse of the lung - collapse is not complete, however, and is prevented from so by three persistent adhesions. These prevent complete obliteration of all the air-containing spaces in the lungs, and this is probably the reason for the persistence of large quantities of sputum.

Further pressure is not indicated as the heart and mediasternum have been pushed to the right side to the extreme limits of comfort.

Later it may be possible to keep lung in its present condition with a smaller pressure.

9.4.19. 400 c.c. air.

23.4.19. E.T. raised for last fortnight.

30.4.19. 200 c.c. air.

14.5.19. A.P. complete.

X-ray report. 4.3.19. Lung well collapsed. Dense adhesion mentioned above still persists, but does not appear to have prevented further collapse.

5.5.19. Lung seems completely collapsed.

Note. Bilateral disease but much more marked on left side.

Copious expectoration + persisted temp. in spite of hygiene and rest absolute - though temp. more unsteady when taken off absol. rest. A.P. adhes. reattempt ^{successful} / - Gradual improvement but not complete because of adhesions, etc.

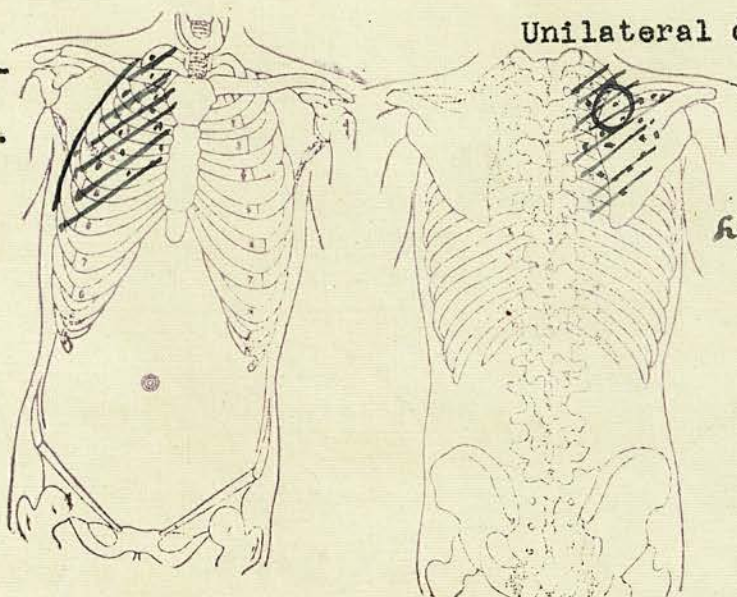
Sug. emphysema and dyspnoea as complic.

Eventually successful and condition improved.

This patient is reported to have improved greatly since.

CASE 3. WINIFRED TAYLOR.

Creps. + +
On inspira-
tion and
expiration.
+ flattening



Unilateral disease.

+ cavity.

Cavernous
B.S., etc.

signs of cavitation

Result:

Much
improved.

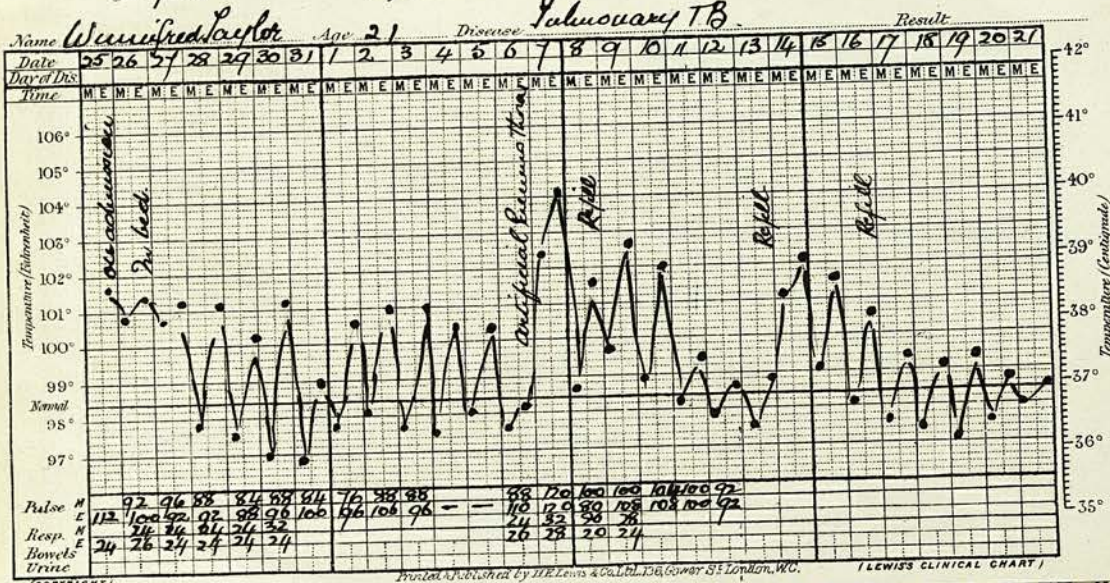
Winifred Taylor. Aet. 21. Adm. 25.8.20.

August.

September.

January TB.

Chart I



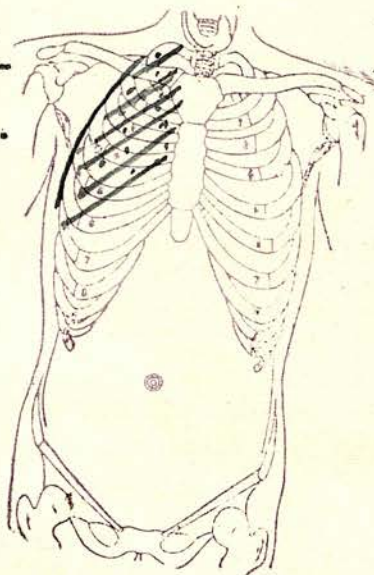
just below 1st rib, where there is an area of hyperresonance.

Ausc.

Creps. present all over rt. lung.

CASE 3. WINIFRED TAYLOR.

Creps. + +
On inspira-
tion and
expiration.
+ flattening



Unilateral disease.

+ cavity.

Cavernous
B.S., etc.

signs of cavitation

Result:

Much
improved.

Winifred Taylor. Aet. 21. Adm. 25.8.20.

Dis. 16.1.21.

Duration of Illness: $1\frac{1}{2}$ years.

T.B. + June 1920.

Symptoms. Cough + Pain +
Emac. + Loss 11 lbs. during last year.
Expect. + Haem. +
Night Sweats + Dyspnoea +

Physical Signs. Inspect. Impaired movement over rt. side chest.

Palpat. " + V.F. + over rt. side.

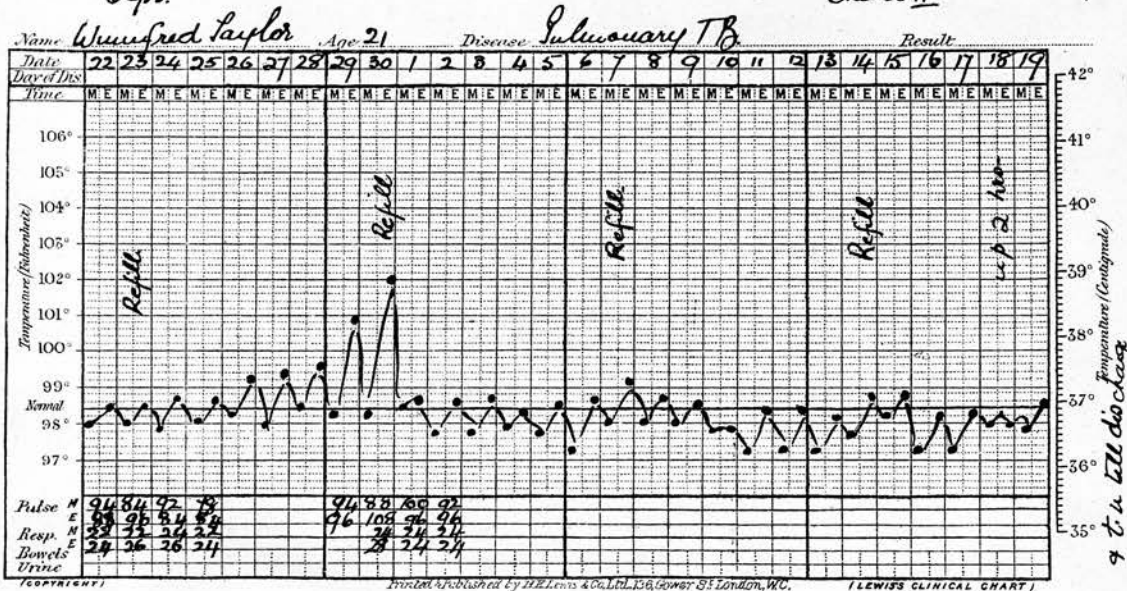
Percuss. Dull all over rt. side chest except just below 1st rib, where there is an area of hyperresonance.

Ausc. Creps. present all over rt. lung.

Over above area V.R. + + to W.P. and B.S. cavernous.

Sept.

Chart II



- Oct. 28. - $\frac{1}{2}$ - 1 \rightarrow + 3 + 4 900 c.c.
- Nov. 11. - 1 - $1\frac{1}{2}$ \rightarrow + 5 + 6 900 c.c.
- Dec. 1. - 1 - $2\frac{1}{2}$ \rightarrow + 4 + 5 900 c.c.
- Dec. 22. 0 - 1 \rightarrow + $3\frac{1}{2}$ + 4 800 c.c.
- Jan. 13. 0 - $\frac{1}{2}$ \rightarrow 3 + $3\frac{1}{2}$ 700 c.c.
- Dec. 28. Good collapse - No adventitious sounds
Dis. O.P. on right side.

Over above area V.R. + + to W.P. and B.S. cavernous.

Sept. 6. A.P. - 4 - 4 \rightarrow 0 - 1 900 c.c. Some
slight surgical emphysema.

Sept. 9. - $2\frac{1}{2}$ - $3\frac{1}{2}$ \rightarrow 0 - $1\frac{1}{2}$ 400 c.c.

Sept. 13. - 2 - $2\frac{1}{2}$ \rightarrow + 1 - 1 500 c.c.

Sept. 16. + $\frac{1}{2}$ - 1 \rightarrow + 1 - $\frac{1}{2}$ 200 c.c.

Sept. 23. - $2\frac{1}{2}$ - 3 \rightarrow + $1\frac{1}{2}$ - $\frac{1}{2}$ 500 c.c.

Sept. 24. No react. after refill. Left lung clear.
R. coll.

Sept. 30. - $\frac{1}{2}$ - $1\frac{1}{2}$ \rightarrow $1\frac{1}{2}$ + 3 600 c.c.

Oct. 7. 0 - 1 \rightarrow 2 + 3 600 c.c.

Oct. 14. - $\frac{1}{2}$ - $1\frac{1}{2}$ \rightarrow + $2\frac{1}{2}$ + $3\frac{1}{2}$ 700 c.c.

Oct. 28. - $\frac{1}{2}$ - 1 \rightarrow + 3 + 4 900 c.c.

Nov. 11. - 1 - $1\frac{1}{2}$ \rightarrow + 5 + 6 900 c.c.

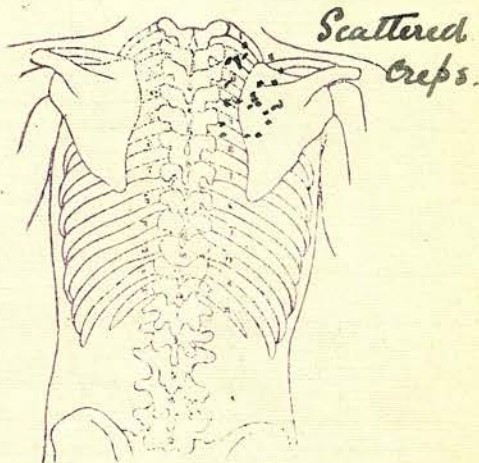
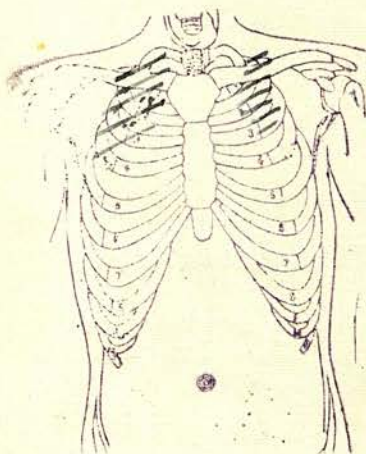
Dec. 1. - 1 - $2\frac{1}{2}$ \rightarrow + 4 + 5 900 c.c.

Dec. 22. 0 - 1 \rightarrow + $3\frac{1}{2}$ + 4 800 c.c.

Jan. 13. 0 - $\frac{1}{2}$ \rightarrow 3 + $3\frac{1}{2}$ 700 c.c.

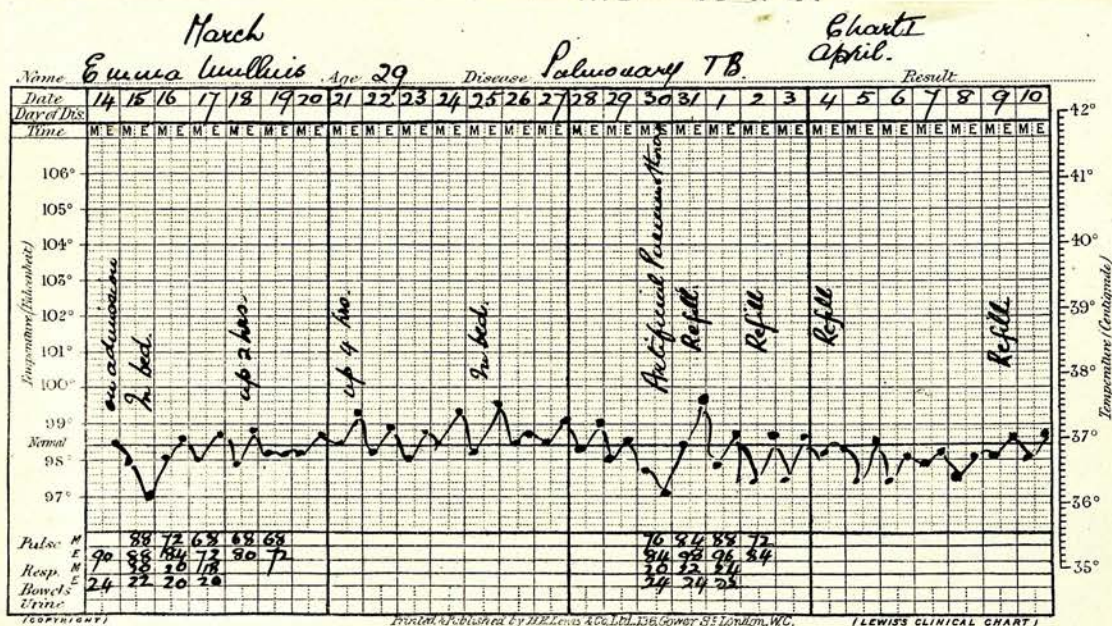
Dec. 28. Good collapse - No adventitious sounds
on right side.
Dis. O.P.

CASE 4. EMMA MULLINS.



Emma Mullins. Aet. 29. Adm. 14.3.21.

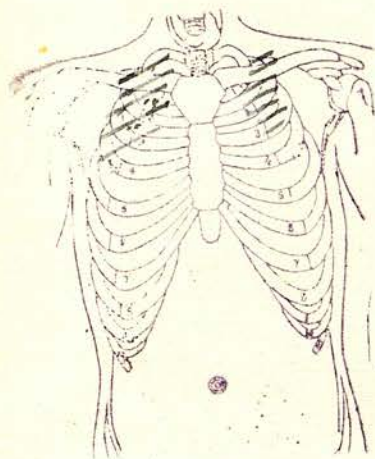
Dis 12 6 21



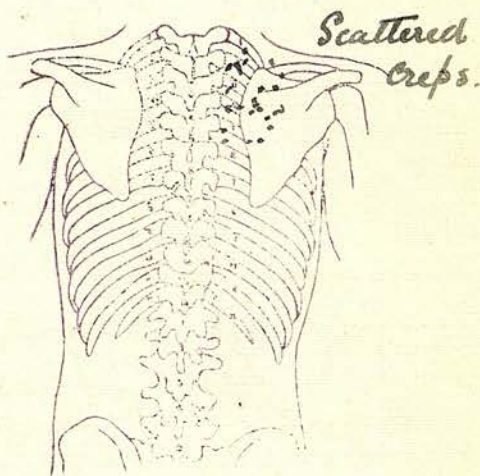
X-ray.

Rt. Dim. transluc. Rt. apex. Patchy opacity rt. upper lobe. Fibrosis with infiltration.

Left. Linear radiating shadows. + No positive evidence of infiltration.

CASE 4. EMMA MULLINS.

B.S. Very harsh.

Emma Mullins. Aet. 29. Adm. 14.3.21.

Dis. 12.6.21.

Duration of Illness. ? 10 years. Acute 5 months.

T.B. + 14.3.21.

History. Hip joint disease at aet. 17. Cough soon after.Symptoms. Cough + Haem. + several large. Night Sweats -

Expect. + Dyspnoea + Emaciation -

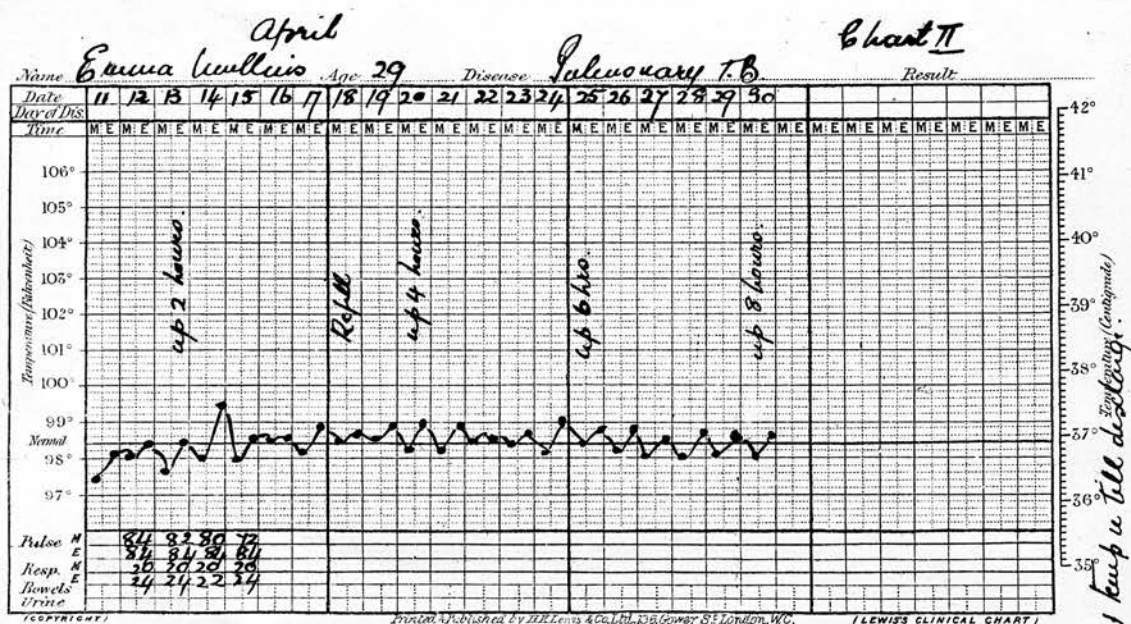
Physical Signs. Inspection. Right side lags behind left.Palpation. do.Percussion. Imp. res. at both apices especially right.Auscultation. At rt. apex.

S. weak + numerous creps. Left apex. B.S. very harsh indeed.

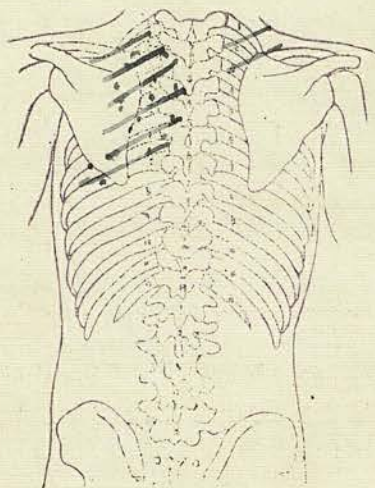
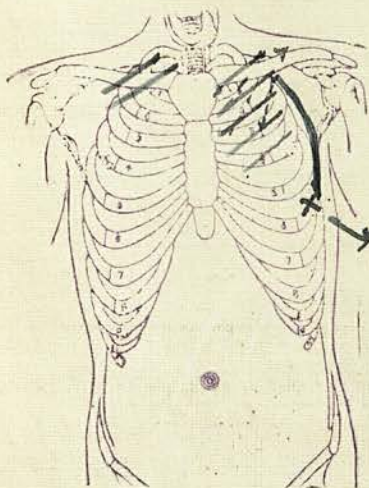
X-ray. Rt. Dim. transluc. Rt. apex. Patchy opacity rt. upper lobe. Fibrosis with infiltration.Left. Linear radiating shadows + No positive evidence of infiltration.

30 March. - $2\frac{1}{2}$ - 5 \rightarrow - $\frac{1}{2}$ - $2\frac{1}{2}$ 450 c.c.
 31 March. - $2\frac{1}{2}$ - $3\frac{1}{2}$ \rightarrow 0 - $1\frac{1}{2}$ 500 c.c.
 2 April. - $1\frac{1}{2}$ - $2\frac{1}{2}$ \rightarrow + $\frac{1}{2}$ - $\frac{1}{2}$ 500 c.c.
 4 April. - $\frac{1}{2}$ - 1 \rightarrow + 2 + $2\frac{1}{2}$ 700 c.c.
 9 April. - $\frac{1}{2}$ - $1\frac{1}{2}$ \rightarrow + 3 + 4 800 c.c.
 13 April. X-ray. Good collapse of rt. lung.
 18 April. - $\frac{1}{2}$ - 1 \rightarrow + 3 + 4 700 c.c.
 2 May. - $\frac{1}{2}$ - $1\frac{1}{2}$ \rightarrow + 3 + 4 800 c.c.
 19 May. - $1\frac{1}{2}$ - $2\frac{1}{2}$ \rightarrow + $2\frac{1}{2}$ + 4 1100 c.c.
 26 May. Good collapse. Adhes. towards axilla.
 Some mottling. ? infiltration of left lung.
 9 June. - 2 - $\frac{1}{2}$ \rightarrow - $2\frac{1}{2}$ - $4\frac{1}{2}$ 1000 c.c.

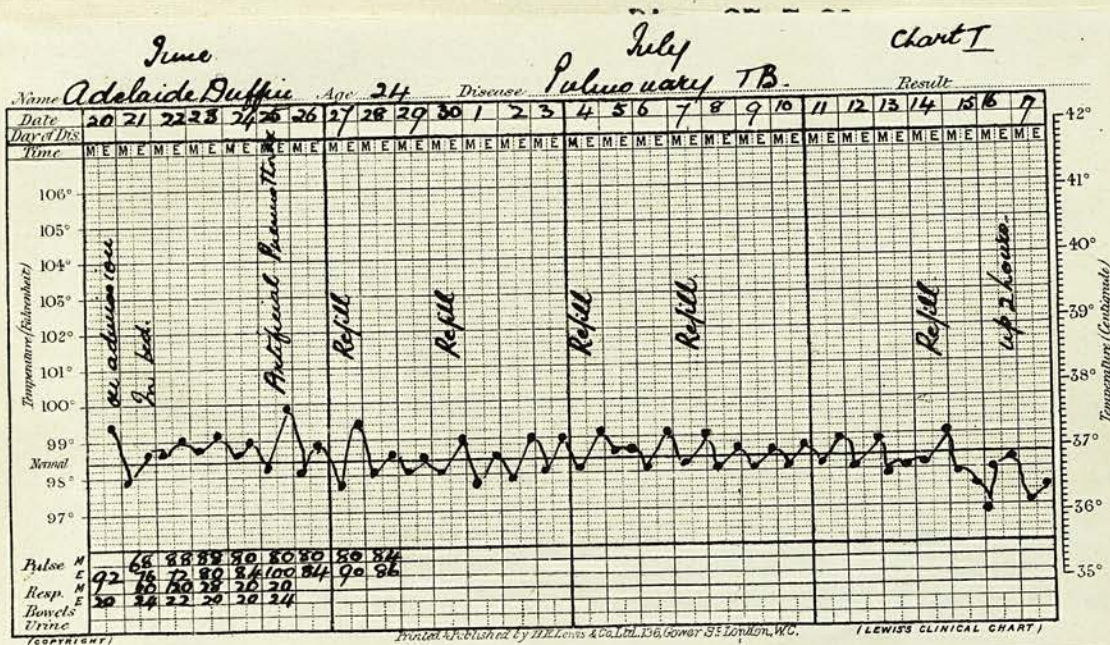
Result: Improved.



CASE 5. ADELAIDE DUFFIN.

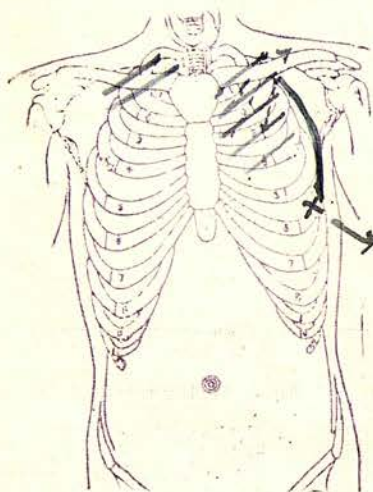


Adelaide Duffin. Aet. 24. Adm. 20.6.1921.

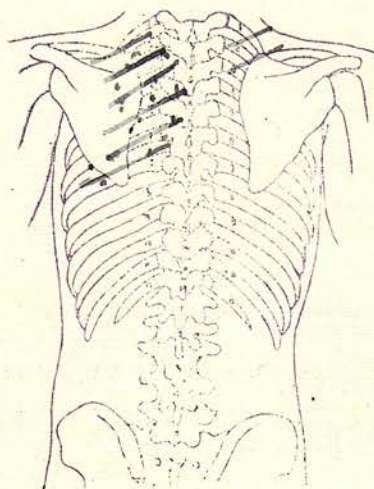


Ausc.

V.R. + at left apex + faint
creps. present posteriorly.

CASE 5. ADELAIDE DUFFIN.

Cardiac
impulse
in ant.
axill.
line.



Almost
entirely
unilateral
disease
progressive.

Adelaide Duffin. Aet. 24. Adm. 20.6.1921.

Dis. 27.7.21.

Duration of Illness: 6 months. T.B. + 22.6.21.

Symptoms. Cough + slight. Pain + in shoulder.

Emac. + Expect. + slight Dyspnoea -

Haemopt. slight one month ago.

Night Sweats +

Physical Signs.

Inspect.

Dim. movement at left apex.
inspection

Palpat.

Confirms /and V.F. + at left side.

Percuss.

Note slightly impaired at rt. apex.
Dullness at left apex.
Ant. and posteriorly extending
over Scapula behind.

Ausc.

V.R. + at left apex + faint
creps. present posteriorly.

25 June. Pneumothorax treatment started.

26 June. - 5 - $5\frac{1}{2}$ → - 2 - $2\frac{1}{2}$ 400 c.c.

27 June. - $5\frac{1}{2}$ - $4\frac{1}{2}$ → - 1 - 2 500 c.c.

30 June. - 3 - $4\frac{1}{2}$ → - $\frac{1}{2}$ + $\frac{1}{2}$ 600 c.c.

4 July. - $2\frac{1}{2}$ - $3\frac{1}{2}$ → + 1 + $1\frac{1}{2}$ 600 c.c.

7 July. - $1\frac{1}{2}$ - $2\frac{1}{2}$ → + $2\frac{1}{2}$ + $3\frac{1}{2}$ 600 c.c.

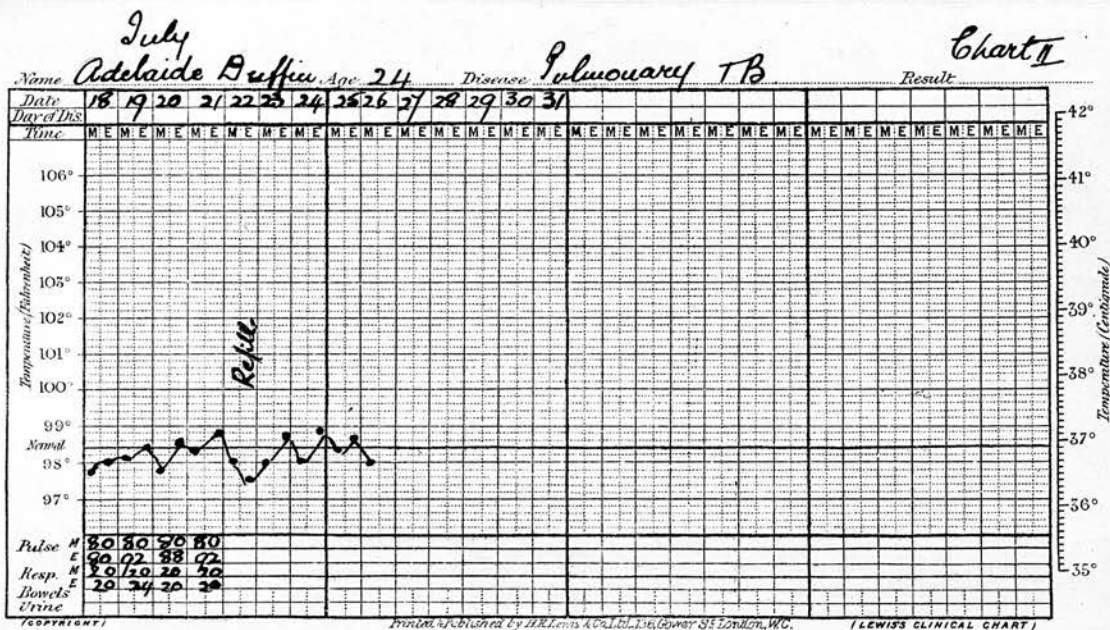
14 July. - $\frac{1}{2}$ - 1 → + $3\frac{1}{2}$ + 4 700 c.c.

July 21. - 1 - $2\frac{1}{2}$ → + $3\frac{1}{2}$ + 4 700 c.c.

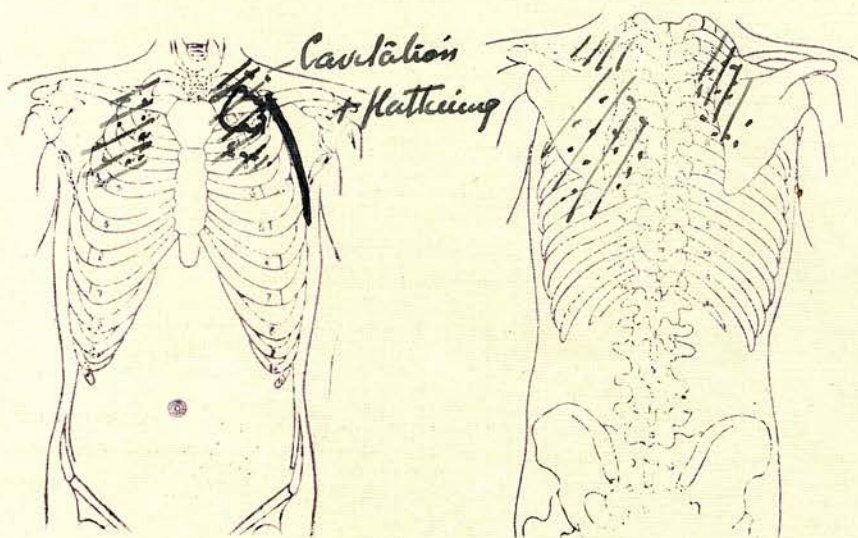
22.6.21. X-ray. Lt. Chest. Heart pulled over to left side.
Infiltration and fibrosis.

Rt. Chest. Compensatory emphysema. Mot-
tling of apex. and bronchial tube
shadows exaggerated. There does not
appear to be extensive infiltration of
rt. lung at present.

Result: Much improved.



CASE 6. WINNIFRED GILL.

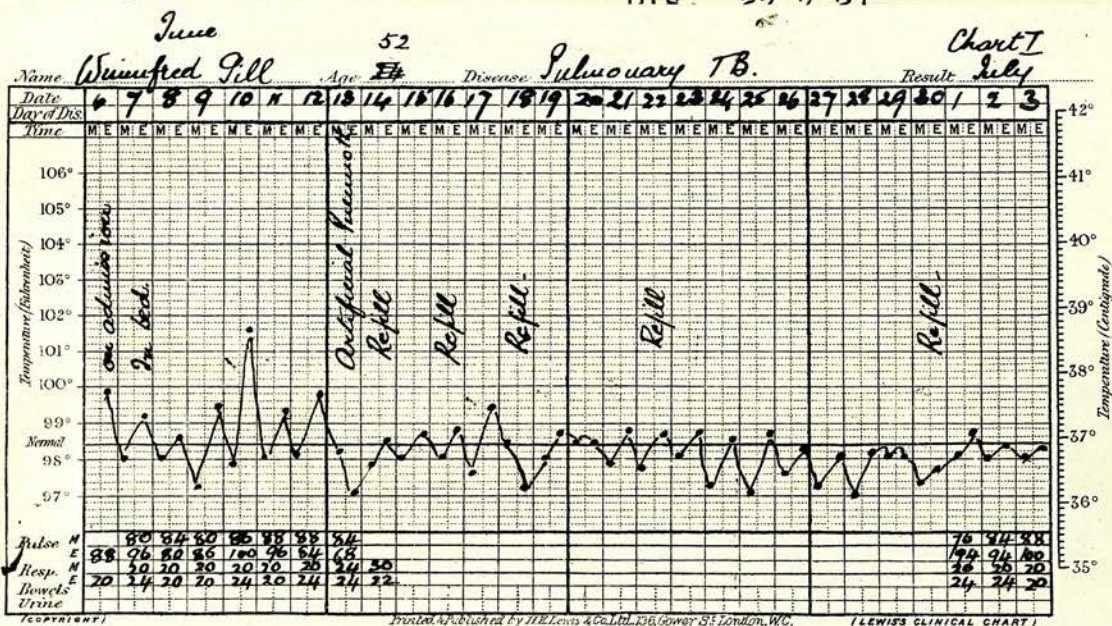


Bilateral
disease.
More
marked on
one side.
Progressive

Result:
Much im-
proved.
Temperature
steady and
gain in
weight.

Winnifred Gill. Aet. 52. Ad. 6.6.21.

Die 27 7 21



y left.

egion
x on

of
resent.

esent.

posteriorly.

X-ray. Heart narrow and vertical and pulled to the left side. Fibrosis and infiltration of left lung except for small area above diaphragm.

Rt. Lung. Infiltration and fibrosis of apex and infraclavicular region.

CASE 6. WINNIFRED GILL.



Bilateral
disease.
More
marked on
one side.
Progressing.

Result:
Much im-
proved.
Temperature
steady and
gain in
weight.

Winnifred Gill. Aet. 52. Ad. 6.6.21.

Dis. 27.7.21.

Duration of Illness: 1 year. T.B. + 8 June 1921.

ptoms. Cough + Emaciation + Haemopt. -
Expect. + Night Sweats + Dyspnoea -
Clubbing +

sical Signs. Slight flattening both apices especially left.

russ. Both apices dull, extending down to scapular region on left side. V.F. + Rt. apex + + left apex on palpation.

2. Rt. V.R. + B.S. weak. Left W.P. and signs of
- Creps. cavity ant. Creps. present.

Post tussive creps. present posteriorly.

X-ray. Heart narrow and vertical and pulled to the left side. Fibrosis and infiltration of left lung except for small area above diaphragm.

Rt. Lung. Infiltration and fibrosis of apex and infraclavicular region.

13.6.21. Pneumothorax treatment started.

13.6.21. - 2 - $3\frac{1}{2}$ → - $\frac{1}{2}$ - $2\frac{1}{2}$ 400 c.c.

14.6.21. - 2 - 3 → 0 - 1 500 c.c.

18.6.21. - $1\frac{1}{2}$ - $2\frac{1}{2}$ → 0 + 1 500 c.c.

21.6.21. - 1 - $\frac{1}{2}$ → + 3 + 4 500 c.c. L. lung
collapsing but
apex still expanded

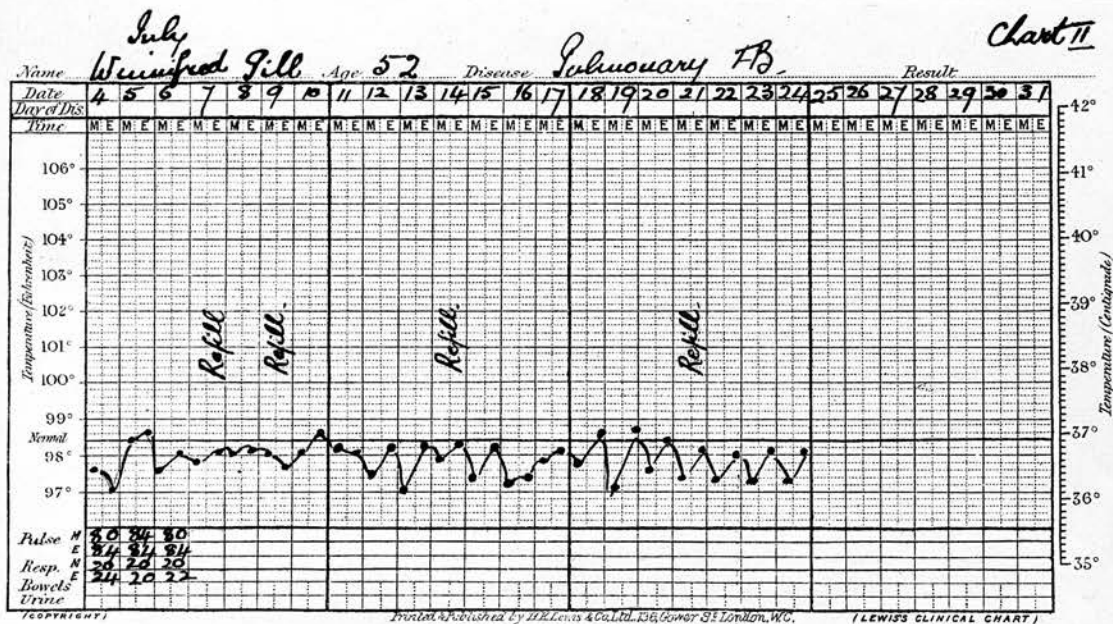
23.6.21. - 0 - 2 → + 3 + 4 400 c.c. Creps.
less at rt. apex.
Left. apex still
uncollapsed.

30.6.21. 0 - 1 → + 4 + $4\frac{1}{2}$ 500 c.c.

7.7.21. 0 - 1 → 4 + $4\frac{1}{2}$ 475 c.c. Left apex
collapsing.

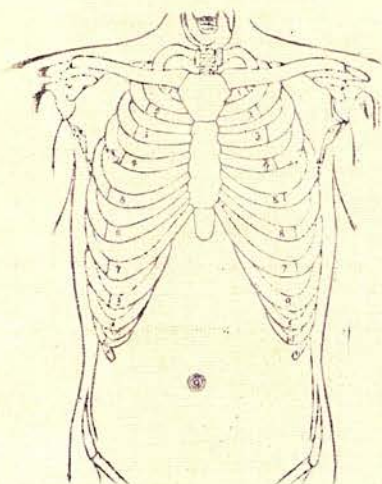
14.7.21. 0 + 1 → $4\frac{1}{2}$ + 5 500 c.c. Gained
4 lbs.

21.7.21. 0 + 1 → $4\frac{1}{2}$ + 5 500 c.c.

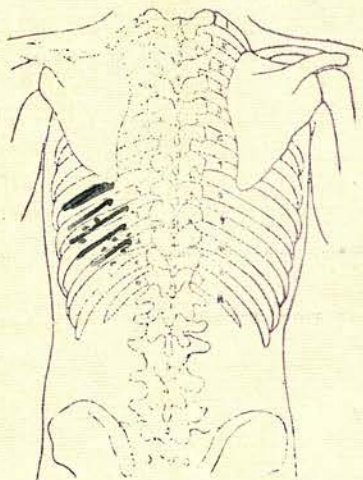


CASE . THOMAS WOODFORD.

Bronchiectasis.



Creps. +
Cav.B.S.
? Dil.
tube



Temperature
pulse &
resp. N.
throughout.
Had creosote intem.
Dis. home
improved.

Thomas Woodford. Aet. 35. Adm. 3.11.20.

Dis. 14.5.21.

Duration of Illness: 9 months. T.B. - 5.11.20, etc.

Symptoms. Cough + Expect. + yellow? Offens.

Night sweats + Pain + left shoulder.

Haem. + streaks. Dyspnoea +

Clubbing + (not marked)

Physical Signs. Slight imp. movement left side.

Palpat. V.R. + left infra-scap. region.

Percuss. Dullness " " "

Ausc. B.S. cavernous + Creps. " "

Progress. Sputum + 1. 1.21 Sputum + but a little less than
at beginning. Wass -

- 10.1.21. Artificial Pneum. (left).
 10.1.21. $\rightarrow - 2\frac{1}{2} - 1$ 600 c.c.
 11.1.21. $- 1\frac{1}{2} - 3 \rightarrow - 1\frac{1}{2} + 1$ 800 c.c.
 13.1.21. $- 1 - 2 \rightarrow 0 + 1$ 700 c.c.
 17.1.21. $- 1 - 3 \rightarrow 0 + 4$ 700 c.c.
 24.1.21. $- 1 - 2 \rightarrow + 4 + 5$ 600 c.c.
 26.1.21. Signs good collapse. Compens.
 harsh B.S. rt. base.
 3.2.21. $0 - 1 \rightarrow 3 + 4$ 800 c.c.
 18.2.21. Signs A.P.
 19.2.21. $- 1 - 2 \rightarrow + 3\frac{1}{2} + 4$ 900 c.c.
 17.3.21. $- \frac{1}{2} - 3\frac{1}{2} \rightarrow + 3\frac{1}{2} + 4$ 1300 c.c.
 14.4.21. $- \frac{1}{2} - 3\frac{1}{2} \rightarrow + 3\frac{1}{2} + 4\frac{1}{2}$ 1100 c.c.
 15.1.21. Good coll. left lung.
 25.1.21. Lung almost completely collapsed.
 17.3.21. Lung well coll. Adhes. 1 diaph.
 13.4.21. Good coll. left lung.

X-ray. 18.11.20. Translucency both apices defective especially rt. apex. Gen. Hypertranslucency of chest. Heart displaced to left. Considerable bronchial fibrosis especially at both bases? Some enlargement of bronchial tube shadows at basis. No infilt. lung tissue seen. ? Early bronchiectasis.

CASE . SAMUEL JENNINGS.

For Haemoptysis.

Result: Haemoptysis stopped. Definite general improvement.

Samuel Jennings. Aet. 34. Adm. 4.4.21.
Dis. 27.7.21.

Duration of Illness: 2 months.

P.B. + 5.4.21.

Symptoms. Cough + Pain + rt. side Night Sweats + prev.
not now.

Expect. + Haem. 3 XV Feb. 21. Dyspnoea +

Physical Signs. Note a little imp. both apices.

Rt. apex. Harsh B.S. - a few post. tussive creps.

Left apex. Harsh, but no adventitious sounds.

CASE . CHARLES RUTLAND.

For Unilateral Disease.

Charles Rutland. Aet. 21. Adm. June 1919.

Dis. to Out Patients Aug., 1919.

Duration of Illness: 3 months. T.B. +

Symptoms. Cough and debility. Temperature 102°F. Pulse 96.

Physical Signs. Extensive signs of infiltration over right upper and lower lobes on left. No râles. Night impairment of note at apex.

June 19.	A.P.	1000 c.c.
June 21.	"	1000 c.c.
June 23.	"	8000 c.c.
June 26.	"	1000 c.c. Temperature now normal and remained so.
July 3.	"	1400 c.c.
July 12.	"	Up gradually.
Aug. 7.	"	1600 c.c.

Dis. Aug. 8th.

Result: Much improved.

CASE . VICTOR BERCHEW.

- For Haemoptysis and progression of disease.

Victor Berchew. Aet. 18. Adm. Nov. 15, 1918. Dis. Nov. 2, 1919.

Duration of Illness: 18 months + T.B. +

Symptoms. Cough + Haem. + on two previous occasions.

Emaciation - progressive.

Physical Signs. Indefinite. Left: Dullness + a few creps. and bronchial breath sounds at left apex. Later a few creps. in left axilla. Right: Not definite. Temp. normal. Pulse 86.

Nov. 22. Sputum coloured. Temp. remains normal, but patient has lost several pounds in weight and physical signs on left side show that disease is extending.

March 12, 1919. Sputum again coloured.

March 26. " " "

April 7. " " "

May 28.	Haemoptysis	(2 oz.)
May 31.	"	(1½)
June 1.	"	Signs more extensive on left side.
June 19.	A.P.	800 c.c.
June 21.	"	1000 c.c.
June 23.	"	800 c.c.
June 26.	"	800 c.c.

Temperature normal but patient continues to lose weight.

2.11.19. Dis. to Home for Advanced Cases.

Result: Disease continued to progress in spite of rest treatment.

CASE . WILLIAM WISE.

For Fever, etc. Disease Unilateral.

William Wise. Aet. 38. Adm. 18.11.18.

Dis. 16.4.19.

Duration of Illness: ? 5 months.

Symptoms. Cough + Haem. + V.

Physical Signs. Infiltration of left upper lobe. Excavation
of left lower lobe. Clear.

Progress. Temperature troublesome, never very high, but
swinging from e.g. 98°F - 99.4° in spite of
absolute rest, etc.

21.1.19. A.P. 500 c.c.

22.1.19. A.P. 500 c.c.

25.1.19. A.P. 200 c.c. X-ray. Left lung
collapsing well.
Some adhesions at apex.

27.1.19. A.P. 900 c.c. T. now normal and
remains so.

29.1.19. A.P. 1000 c.c.
 3.2.19. A.P. 600 c.c.
 7.2.19. A.P. 800 c.c.
 13.2.19. Up 2 hours.
 17.2.19. A.P. 800 c.c.
 5.3.19. A.P. 800 c.c.
 20.3.19. A.P. 1200 c.c.
 14.4.19. A.P. 1000 c.c.
 16.4.19. Dis. to Out Patients. To attend for
 refills.
 23.2.20. Admitted for a short time owing to the
 formation of fluid.
 25.2.20. Aspirated.
 1.3.20. X-ray shows no fluid present.
 10.3.20. A.P. induced.
 20.3.20. Discharged home. Patient attends O.P.
 every six weeks or so for refills. Is
 at work. No cough or sputum.

Physical signs. Show that fibrosis has occurred and that
 the heart is drawn towards the left side.

Result: Much improved. Temperature steadied. Although a
 pleural effusion occurred, treatment could be carried on, and
 resulted in fibrosis and healing to such an extent that
 patient was able to return to work.

CASE . FRANK RIDER.

For Fever, etc.

Frank Rider. Aet. 26. Adm. 23.4.19.

Dis. 13.3.20.

History. March 1919 had two haemoptysis of large amount.

T.B. +

Physical Signs. Infiltration of right upper and lower lobes.

Dull Bronchial Breath Sounds and whispering pectoriloquy.

Progress. Temp. except on "absolute rest" always unsteady, swinging up to 99.4°, etc., this cont. until Aug. 19.

7.8.19. A.P. 1200 c.c.

9.8.19. A.P. 1200 c.c.

11.8.19. A.P. 1200 c.c.

16.8.19. A.P. 1200 c.c.

Effect so far not good, temperature was on the whole rather higher than before.

6.9.19. A.P. 1200 c.c.

20.9.19. 1200 c.c.

9.10.19. Temp. rose suddenly to 104°F, and remained up for 12 days, after which it dropped to 99°F. No change in Physical Signs.

27.10.19. A.P. 800 c.c. X-ray shows presence of fluid to 5th interspace and adhesions preventing complete collapse of lung. Temp. remains normal at rest.

11.12.20. Refill. Patient up 4 hrs. Temp. remained normal.

19.2.20. Refill and some fluid withdrawn. Temp. rose to 103°F, and remained ranging to about 99.2°.

13.3.20. Discharged. Downs Sanatorium in statu quo.

Result: Condition much as on admission. Some temp. improvement, but possibly lasting improvement was due to adhesions preventing complete collapse of lung.

CASE . FRANK OSBORNE.

For Haemoptysis + fever.

Frank Osborne. Aet. 22. Adm. 14.5.19. Dis. 8.4.20.

Symptoms. Cough + Haemoptysis + on 2 occasions, 2 months and again 3 weeks ago.

Physical Signs. Left Apex. Note impaired on percussion.

Bronchial breath sounds. No added sounds.

Temperature - steady on admission.

June 11. Haemoptysis.

June 14. " Temperature now 99.4°
and remains elevated
above 99°F until end
of July.

Aug. 5. Coloured sputum. Temp. shows no signs
of settling.

Sept. 29. A.P. 900 c.c. left side.

Oct. 1. A.P. 1100 c.c.

Oct. 4. A.P. 1100 c.c.

Oct. 11. A.P. 1000 c.c.

Oct. 25. A.P. 1100 c.c.

Nov. 17. A.P. 1400 c.c.

Dec. 11. A.P. 1800 c.c.

Jan. 8. A.P. 1400 c.c.

Feb. 5. A.P. 1500 c.c.

These injections had no effect at all on the temperature which still remained unsteady and above 99°F.

X-ray shows marked adhesions in the region of the axilla.

No evidence of presence of fluid or of efficient collapse.

March 3. Air in pleura nearly absorbed. Evidence of marked increase in disease. Creps. present all over both upper and lower lobes of left lung.

April 8. Dis. home.

Result: No improvement. Some progression of disease.

Adhesions evidently practically prevented induction of

Artificial Pneumothorax.

CASE . CHARLES THESIGER.

For Recurrent Haemoptysis.

Charles Thesiger. Adm. August 1919. Dis. December 1919.

History. 1914. Haem. X and repeated six weeks later.

June 1915 - April, 1918, at work as motor mechanic.

April, 1918. Haemoptysis - was at Frimley Sanatorium until November 1918.

August, 1919. 4 attacks of haemoptysis and one on night of admission.

On admission. For Haemop. T. 101, P. 100, T.B. +

Physical Signs. Infilt. left upper and lower lobes. Right lung clear.

1.9.19. A.P. 800 c.c. Same evening haemoptysis vii

3.9.19. Haemoptysis iv. A.P. 1200 c.c.

6.9.19. A.P. 1200 c.c.

10.9.19. A.P. 1200 c.c.

18.9.19. A.P. 1000 c.c.

2.10.19. A.P. 1200 c.c.

24.10.19. A.P. 1500 c.c. Allowed up now for 4
hours daily.

22.11.19. A.P. 1000 c.c.

X-ray. Left lung collapsing well. Heart pushed over to
right.

Dec. 19. Discharged.

Result: Much improved. Attends for refills.

CASE . EDWARD GOSLING.

For Unilateral Disease.

Edward Gosling. Aet. 30. Adm. 15.9.19.

Dis. 12.12.19.

Symptoms. Cough, etc. Haemoptysis: Dec., 1918.

March, 1919.

August, 1919.

On Admission. Temperature normal, Pulse 70.

Physical Signs. Infilt. left upper and lower lobes.
Right lung clear.

Nov. 10. A.P. 800 c.c.

Nov. 12. A.P. 900 c.c.

Nov. 17. A.P. 1200 c.c.

Nov. 24. A.P. 1000 c.c.

Dec. 8. A.P. 1000 c.c.,

X-ray. Shows good collapse of left lung.

Discharged December 12, 1919. Attends for refills.

17.2.21. Lung completely collapsed. Hydropneumothorax to level of 4th interspace.

Result: Improved.

CASE . CECIL TERRY.

For Haemoptysis.

Cecil Terry. Aet. 43. Admitted 27.11.18.

Discharged 8.8.19.

Symptoms, etc. Cough + T. normal. Pulse 90. T.B. +

Haemoptysis: $\frac{3}{4}$ pint. 6 months ago.

$\frac{1}{4}$ " 4 " "

$\frac{1}{4}$ " 3 " "

and several since.

Physical Signs. Infiltration, dullness and râles over right upper and lower lobes. Left lung clear.

Sputum. Tinged. Remained so, on and off, for a period of 2 - 3 months, although on "absolute rest".

5.3.19. A.P. 500 c.c.
 6.3.19. A.P. 1000 c.c.
 10.3.19. A.P. 700 c.c.
 13.3.19. A.P. 1200 c.c. + 5 c.c.
 17.3.19. 600 c.c. + 3.
 20.3.19. 1000 c.c.
 31.3.19. 1000 c.c.
 14.4.19. 1000 c.c.

Was gradually allowed up and by June 5, 1919, he was up 8 hours. Towards end of June sputum again became slightly coloured. This soon cleared up and he was discharged early in July, but small haemoptysis persisted, so he was readmitted October, 1919.

Physical signs entirely right-sided as before. Had three refills, and was discharged improved. Now attends for refills.

Result: Improved.

CASE . CHRISTOPHER HUNTINGDON.

For Bilateral Disease, more marked on one side and extending.

Christopher Huntingdon. Aet. 50. Adm. 6.8.19.

Died 4.2.20.

History: Pleural effus. October 1918. Cough since.
Haemoptysis in July, 1919. T.B. + in sputum.
Temperature 100. Pulse 102.

Physical Signs. Infilt. with dullness and râles right upper and lower lobes and left upper lobe.

2.10.19. Signs extending on right side.
A.P. 800 c.c. Adhesions present.

4.10.19. A.P. 1400 c.c. X-ray. Lung not collapsing well.

20.10.19. Lung collapsing but evidence of dense adhesions above and below collapsed area.

25.10.19. 600 c.c.

20.11.19. 1000 c.c.

4.2.20. Died.

Result: Died.

CASE . NEVILLE PRENTICE.

For Haemoptysis, etc. Example of open
(Surgical) Pneumothorax.

Neville Prentice. Aet. 32. Adm. 28.11.19.

Died 5.3.20.

History. Haemoptysis. June 1918, following trauma.

March 19. Haemopt. + cough + and since then has had
repeated haemoptysis. T.B. +

On admission: T. 99.6.

Physical Signs. Infilt., dullness, creps. at left upper lobe.
Dullness at apex.

Nov. 29. Haem.

Nov. 30. "

Dec. 1. "

Dec. 4. A.P. 1000 c.c.

Dec. 6. A.P. 1000 c.c. Temp. falls to normal.

Dec. 8. A.P. 950 c.c.
Dec. 15. 1000 c.c. Temp. still normal.
Jan. 1. 1000 c.c. X-ray, lung not collapsing
well. Dense adhesions
at apex.
Jan. 15. 400 c.c.
Jan. 21. Getting up.
Jan. 29. 1000 c.c.
Feb. 11. 800 c.c.
Feb. 16. 1000 c.c. X-ray.
March 5. Operat. attempt to divide adhesions.
Death from haemorrhage.

Result: Death.

CASE . ALICE BEST.

For fever, etc.

Alice Best. Adm. 23.6.19. Dis. 19.12.19.

Symptoms. Cough + Emaciation - Sputum + Haemop. -
Amenorrhoea.

Temp. 103. P. 136. And remains swinging until August 9th.

Physical Signs. Infilt., dullness + cavitation, and creps.
right upper and infiltration right lower
lobe posteriorly.

Aug. 9. A.P. 1200 c.c.

Aug. 11. A.P. 1200 c.c. Temp. still rises to 101°
in evening.

Aug. 16. A.P. 800 c.c.

Aug. 25. A.P. 1300 c.c. Temp. falls to range
between 99 and 100.

By Sept. 6. Temp. normal and remained so.

Sept. 8. A.P. 1000 c.c.

Sept. 20. 800 c.c. X-ray. Lung collapsing well,
but adhesions present
at apex.

Oct. 17. 800 c.c. Getting up.

Nov. 17. 800 c.c.

Dec. 15. 700 c.c. X-ray shows good collapse.

Dec. 19. Discharged much improved. Has gained
nearly a stone in weight. Attends O.P.
for refills.

Result: Much improved.

CASE . LILLIAN VARNEY.

For swinging temperature, etc.

Lillian Varney. Aet. 31. Adm. 15.10.19.

Died 5.2.20.

Symptoms. Haem. + April 19. Cough + T.B. + T.102°F.

P. 115. R. 28. T. remained high and swinging.

Physical Signs. Infiltration right upper and lower lobes
with probably some excavation of right
upper lobe. Left lung clear.

Nov. 6. A.P. 1000 c.c.

Nov. 8. A.P. 800 c.c.

Nov. 12. A.P. 1000 c.c.

Nov. 20. A.P. 900 c.c. No marked change in
temperature.

Dec. 4. . A.P. 1000 c.c. T. commences to fall gradually.

X-ray. Rt. lung appears completely collapsed.

Dec. 26. T. steadier, ranging from normal in morning to 99.6 in evening.

Jan. 1. A.P. 700 c.c.
1920.

Jan. 8. A.P. 700 c.c.

Jan. 14. A.P. 800 c.c.

Jan. 18. Temp. normal and remained so until death Feb. 5 from T.B. Enteritis, shown post mortem.

Result: Death from T.B. elsewhere.

CASE . GEORGE DOLBY.

For Unilateral Disease. Extending.

George Dolby. Aet. 30. Adm. 12.1.20.

Dis. 4.5.20.

Symptoms. Cough + T.B. + in sputum.

Physical Signs. Infiltration upper and lower lobe on left side. Right lung clear.

1.3.20. Evidence of considerable extension in left lower lobe. Temp. unsteady - 98-99.8.

25.3.20. A.P. 800 c.c.

27.3.20. A.P. 1200 c.c.

30.3.20. A.P. 1400 c.c.

8.4.20. A.P. 1200 c.c. Up 2 hours. Temp. steady.

22.4.20. Up 6 hours.

4.5.20. Discharged. O.P. for refills.

Result: General condition much improved. Disease apparently arrested.

ALICE BLOOMFIELD, M.B., Ch.

THESIS for M.D.

ARTIFICIAL PNEUMOTHORAX IN THE TREATMENT OF PULMONARY
TUBERCULOSIS.

With Notes on 20 Cases so Treated.